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ON THE EXCITABLE AREA OF THE CORTEX, AND ITS RELATIONS TO THE COLUMNS OF THE SPINAL CORD.

A Reply to Professor HORSLEY.

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FIFTEEN years ago, when repeating the experiments of Hitzig and Fritsch on the excitable area of the cerebral cortex, I found some new properties of this cortex, and gave a new explanation of the well-known results of its electric excitation. These facts, previously announced in the 'Giornale Imparziale Medico' of Florence, were described first in the appendix to the second edition of my 'Lezioni di Fisiologia Sperimentale,' Florence, 1873, p. 523–540, and later in the first part of the 'Rivista Sperimentale di Frenatria,' for 1876, and in Pflüger's 'Archiv f. Physiologie,' vol. xxx. (1883) pp. 212–275, and in the same journal, vol. xxxiii. (1884) pp. 264–271.

I cannot repeat all, nor even the principal part, of what I said in the above quoted papers; but it seems to me that Prof. Horsley, before undertaking the criticism of my views on the subject, should have made himself thoroughly acquainted with these memoirs, where, I believe, I have plainly justified my opinions. He should not have directed his criticism against a single paper by Dr. Huggard, that was not intended to demonstrate the reality of my theories, but merely to record

some facts, which had struck the author whilst working in my laboratory.

I encouraged Dr. Huggard to publish his paper, not only because it contains some facts which could not yet be known to English readers, but also because I hoped that it would draw attention to my former publications, where the views to which he alluded have been justified by experiments, and in which my experimental methods are sufficiently set forth. If Prof. Horsley, after having read and repeated my experiments, still retains some doubt as to the reality of the facts, or to the validity of the conclusions, I am ready to enter upon a discussion with him, and to correct the errors that he can prove I committed, either in my method or in my reasoning.

But for the present I can only restate the most general results of my researches, giving merely one or two examples of the facts that have determined my judgment.

Symptoms of the complete destruction of the Excitable Area.

All the experiments I shall mention were made on the dog, when no other animal is specially indicated. The conclusions therefrom refer to the dog. In researches on the central nervous system, I never venture to generalise results obtained on one or two species of animals only. For other animals, such results point to possibilities which we must keep in view, but cannot take as realities, unless confirmed by special experiments. Hence I trust the reader will be able to accept the results, without being prejudiced by clinical or experimental facts, resulting from observations made on higher animals.

When we endeavour in a series of dogs to effect the destruction of all the excitable cortical area, we never can make in every case the conditions so exactly alike, that absolutely the same result is obtained in the early days after the operation. At this period traumatic irritation still exists; but after we have reached the stage which I have called "traumatic equilibrium," we distinguish certain symptoms which are uniformly present *in all the cases*; whilst others are shown only by animals with a deeper, more extended destruction of the cortical and a part of the white substance.

So we must distinguish traumatic from permanent symptoms; and among the permanent, those which are essential and general from those which, accompanying the essential ones, are present only in a certain number of cases.

I shall not enter here into an enumeration of the traumatic symptoms, observed before the so-called "equilibrium" has been reached; it may be sufficient to say, that in the first period the essential symptoms are not wanting, but they are complicated by others, which disappear after a certain time, varying from some hours to three weeks. It is evident that, contrarily to the views expressed by some authors, these do not depend upon the destroyed organs, but upon others, which are *probably* in the proximity of the wound. I take it for granted that the function of a destroyed organ is lost for ever, and cannot be re-established by compensation. It is not the loss of a function, but only the secondary effect of that loss, that can be diminished by the exercise of other organs. Hence we must expect that the true function of the cortex is lost for ever after its destruction, and that all such symptoms as disappear, either wholly or in part, are due *not to the loss of the organ*, but to the effect of traumatism. I must insist on this principle, which has been very often forgotten in the discussions on the functions of the brain.

Among the symptoms that disappear in the early period, sometimes within the first hour, after the operation, after the dog has recovered from the ether narcosis, is a diminution of all the sensations in the limbs, produced by pricking with a needle, by pressure, or by traction of the muscles. These sensibilities are not entirely lost (as sensibility for touch and cold really is), but they are feebler than in the normal state, feebler than on the corresponding parts of the other side of the body, even when on this other side there is no hyperæsthesia.

This state of the sensation (tactile anæsthesia) produces, on the side opposite to the operation, a surprising indifference to the positions of the limbs. This indifference has sometimes been taken for an expression of paralysis or paresis. It is much more evident when the dog is at rest, there being no motor current; here it cannot be *produced* by the weakness of this motor nerve-current. When the dog is walking, he shows

in a certain degree, the appearance which I have described on page 143 of my 'Physiology of the Nervous System' (Jahr. 1858–59), as the effect of the section of the posterior spinal roots, with perfect conservation of motor energy. And these observations, made first at Berne in 1856 and 1857, have been repeated and confirmed within the last few months by Dario Baldi[1] in a very interesting series of experiments, in which he was able to preserve for a very long time the dogs that had been operated upon. It is singular that this author, who arrives at no new conclusions, does not mention the experiments of Panizza, nor of Stilling, nor of myself, which have since been repeatedly demonstrated in the laboratory of Florence, where Baldi himself is now working.

When we stretch a limb, or bring it in a very inconvenient position, the animal—unless the equilibrium of its body be disturbed—does not resume its attitude before walking or making a general movement. Can this dog make a voluntary movement with a single limb, unless under the influence of the performance of a combined movement due to a spinal mechanism, like that of walking?

If I can prove that an intentional isolated movement is still possible, the want of such a movement in the case we consider here, can only be attributed to the want of intention. And the absence of intention can only be due to the fact, that the tactile insensibility, the existence of which is evident from other experiments, hinders the dog from being aware of the irregular position of his limbs.

A dog in which the state I am now describing lasted for six hours, was lying on the floor with the anterior left extremity stretched out at right angles with the body. I brought a metallic electrode, connected with a small Ruhmkorff coil, near the ball of the foot, and discharged three times into it a unipolar spark. After every discharge the foot was removed and brought forward, and hidden under the neck of the dog. I replaced the limb in its former stretched position, where it remained quiet till the following discharge. At the fourth time, the animal, seeing the movement of my hand and the electrode, did not wait till I was close to the foot, but before

[1] Baldi, 'Lo Sperimentale;' Florence, 1885.

the discharge occurred, removed the limb to hide it under his neck. Thus it is clear that a voluntary movement, a movement due to a mental impression, and not to the action of a general mechanism, was possible. It is exceptional that, as in this case, a cerebral movement occurs so early; but this exception is not a very rare one. I must remark, that the local impression which produced here a movement by a simple recollection was not of an extraordinary strength, because the dog was not put to flight, nor to a general movement; while the latter effect could easily be produced by firmly pinching the tail with my fingers. The phenomenon persisted for some weeks, till the same dog could almost give the paw when ordered.

The indifference to position is but the effect of insensibility.—When at a later period this insensibility is more and more reduced to want of sensibility for touch and for cold only, the indifference is less marked, though it persists indefinitely for those abnormal positions which the normal dog perceives only through the sensation of touch. And in the same manner the want of movement is reduced to those movements, which are normally excited by touch or by cold.

When the traumatic equilibrium sets in, the former are very quick, the latter become slower and slower. But before and during the return not a single muscle is paralysed, nor a single movement impossible. This is a point in which even Goltz and Munk, who were not always of this opinion, have for some years agreed perfectly with my observations. Lautenbach, who had assisted at some of my experiments, has published a paper in order to defend the same opinion. I must insist on the fact, that most of the dogs in which all the excitable cortex has been completely removed, begin after some weeks to use their anterior extremities as a hand. They give the paw at our request, if they have formerly been taught the trick. They hold a bone or a hard piece of bread, or rather they try again to hold it with the two anterior extremities. Cats, squirrels, and rats climb with the aid of the two or four extremities, and they place them, not as if they were directed by a pre-established mechanism, but in accordance to information given by visual and by central impressions generally. I have already insisted on all these points in my

first-quoted paper in Pflüger's 'Archiv,' where I have explained the remaining imperfections of these intentional movements by the loss of the direction given by the different organs of tactile sensibility. By reading this paper, Prof. Horsley may easily be convinced that the hiatuses he speaks of ('BRAIN,' *l. c.*, p. 45) exist less in my description than in what he knows of my observations. Most of the characteristic features with which Prof. Horsley tries to fill up these hiatuses must be again put out of the way, if we wish to know the real facts, and not Horsley's ideas about the facts.

It is true that in some, but far from all, cases, certain intentional and isolated movements of the fore and even of the posterior limb, did not return after several months. These are the cases in which I think (see Pflüger's 'Archiv,' 1883) that not only the cortex, which is only a channel for tactile (and thermic) impressions, but that the *centre* for these sensations, has also been damaged. I assume here a greater extension of the extirpation, though I must say that post-mortem examinations have not yet justified this opinion. I have found cases of this second category, in which the wound examined with the naked eye was only some millimetres deep, and have seen two examples of the first category, in which intentional movement returned, and in which the wound extended deeply into the brain, so as to destroy the upper part of the lateral ventricle. These examples prove, that the *loss of intentional movements is not one of the essential effects* of the complete destruction of the excitable area of the cortex.

Though the dog uses his anterior extremities for holding the food, he does not use them well. At first he makes the suitable movements to touch or to fix the bone. But while he is eating he must be guided by touch, in order to hold fast when he makes traction with the teeth. Our dog cannot feel the traction; he cannot feel whether he touches or not, and after a short time, still keeping his fore-paws fixed, he touches no more. The flexion of the toes, not being limited by the resistance of the object, becomes exaggerated, the paw is bent and kept in flexion. After a while we see the paw held in the same position, but at a certain distance from the object, the dog not being aware that he is no longer

touching it. Strength is not wanting, nay, at the beginning of the movement is sometimes *stronger* with the insensible paw; as can be shown in dogs, and better still in squirrels and cats, by offering them a piece of dry or half-dry bread hanging on a string. The impressions of the toes and claws of the insensible side, which cannot hold fast, are often more visible and deeper than the impressions of the other side.

As I said as early as 1858, in my 'Physiology of the Nervous System,' the movements which are not guided by sensibility are as often too strong as they are too weak. The same rule holds good for the strength of the movements with which the toes are placed on the ground.

It is more than ten years since I investigated this point by moistening the under side of the foot with oil, and observing the prints left by the animal walking along my long corridor in Florence. The sound produced by the contact may, on a resounding ground, give the same evidence in dogs, and still better in squirrels.[1]

When a dog or a cat is shaking itself (for instance, after having been sprinkled with water), the insensible extremities cannot quickly or adequately moderate and regulate the contact with the ground; they slip, and the animal may fall on the same side. On this point we must correct what Prof. Horsley (*l. c.*, p. 45) says of the position of our dogs that have been operated upon.

Prof. Horsley believes he must draw my attention to the position of the toes. But the possibility of making the dog or the cat stand with the toes of the anæsthetic side doubled up, so that they really stand, and somehow walk, on the dorsum of the foot, has been for me for a long time[2] the simplest and most evident sign of anæsthesia of touch. For many years I used to make my students try for themselves to double up the toes of the two sides, and feel the difference in the resistance. But I must add to what has been said by Prof. Horsley, that, when the dog has returned to perfect traumatic equilibrium, he no longer, or very seldom, takes this position by himself; like an ataxic man, he accustoms himself

[1] See Pflüger's 'Archiv,' 1883, p. 435.

[2] See my 'Lezioni di Fisiologia,' 2nd edition; Florence, 1873, p. 537.

to a stronger tension of the muscles, the movements of which he no longer feels.[1] But we can produce the aforesaid position with our hands, when the dog is standing quietly on the table, and observe the resistance of the dog, and the persistence of the position. I believe I have clearly and sufficiently proved[2] that the non-resistance of the dog to this tactile excitation does not depend, as Prof. Horsley still believes, on a paralytic state, and that there exists no trace of paralysis, either in the extremities or in other muscles of the body.

We can prove in an analogous manner, as I did it in the last quoted paper for the toes, the non-existence of paralysis and the existence of tactile anæsthesia in one half of the head, opposite to the decortication of the brain. This has been shortly indicated, as early as 1873, in my 'Lezioni di Fisiologia,' p. 538.

Should I repeat here all that I have published on that matter, this paper would assume the length of a large volume; and such a repetition would be useless. I believe I have said enough to convince Prof. Horsley, that he must study the original articles and those of Goltz and Munk, if he wishes to have an idea of the actual state of our knowledge on this subject. As I said in the beginning, Dr. Huggard's valuable articles (and Prof. Horsley seems to know only these communications, which are and must be fragmentary) contain only the indication of some new facts, which must be misunderstood when all the premises are wanting.

I have spoken of Goltz, and I must add that this author, who now adopts nearly completely my explanation of some facts, holds that tactile sensibility is not completely wanting after decortication of the excitable area, but that it becomes much enfeebled. I will not enter here on the discussion of this question, which has for us only a secondary interest, and cannot change our explanation of the symptoms. Even though a trifling remnant of tactile sensibility still remains, it is not sufficient to provoke a motor reaction in the ordinary state of the animal.[3]

[1] See the outline figure of an ataxic cat in my paper in Pflüger's 'Archiv,' 1883, p. 275, Fig. 2.

[2] Pflüger's 'Archiv,' 1883, p. 264 et seq.

[3] Ibid. vol. xxxiv. p. 465.

Already in my paper of 1876 in the 'Rivista di Frenatria,' I remarked that a dog under the influence of strychnia, seems to be nearly equally excitable to tactile impressions on both sides, though on one side he has been deprived of the cortical excitable area. It is true he is excitable, but does he receive from both sides well-localised impressions? Some facts I have witnessed lead me to doubt that he knows that he has been touched on the anæsthetic side.

All I have said till now relates to full-grown or nearly full-grown animals. Is it the same for very young dogs? New experiments must be made on this subject. But I must add that in two instances, among more than 200 experiments, the state I suppose to exist in new-born animals was still existing in the latter part of the first year. And this *on one side only*. The other side of the brain showed the ordinary effect of decortication.[1]

All these facts, and those observed and analysed by other physiologists, justify the conclusions:

(1.) That the direct and only effect of the ablation of the excitable area in fully grown dogs and many other animals is tactile anæsthesia as observed by me, and insensibility for cold as first observed by Herzen.

(2.) That this effect persists without diminution as long as the animals live or can be observed.

(3.) That the simple and the co-ordinated muscular movements, or muscular innervations, are not involved so as to become either feeble or paralysed.

(4.) That some defect or some hesitation in the ordinary movements may arise in variable intensity through want of tactile sensation.

Such a state cannot be called paralytic, as Prof. Horsley would still call it. Seeking a name for it, the expression *Ataxy* presented itself very obviously. If I was wrong in adopting it (and Herzen followed me in that course, and Wernicke seems to approve of it), Prof. Horsley may propose a better one, and I shall accept it.

[1] See for one of these cases the paper of Herzen and Löwenthal, in one of the forthcoming numbers of the 'Revue Zoologique Suisse.' The other is still living in my laboratory, and will eventually be examined post-mortem by me or my friend, Prof. N. Löwenthal.

But in the meanwhile it seems to me that we must preserve it, unless we wish to create a new one. Thus the position of the toes remains for us the best and most expressive sign of ataxy. And a dog which has lost the excitable area is not only ataxic, but appears to me, in the words of Homer:

πόλλον ἀτάξιος ἄλλων.

I have endeavoured to find somewhere on the surface of the cortex a true motor centre. I have not found one. Even after a complete section of the internal capsule of one side, there exists no motor paralysis.

Influence of the Posterior Columns of the Spinal Cord on the Excitable Area of the Brain.

We see from the preceding exposition that, from the beginning of my experiments, the analysis of the facts led me to the conclusion, that the pretended motor centre does not exist in dogs, and that it must really be called a sensory centre, if it is a centre at all, and not, as I believe, the most central patch of afferent channels.

A new experiment I made some years ago led me to the conclusion, that what I have considered to be an analogy between the effects of decortication of the brain, and of division of the posterior columns of the spinal cord, is more than a mere analogy, that it is identity. The fifth day after the division of the posterior columns of the cord, the excitable area of the brain loses its influence, and no longer gives rise to reflex actions in the parts of the body posterior to the division of the posterior column.

Prof. Horsley, without being acquainted with our method of experimenting, believes that he has repeated our experiments. His results were in general negative, or nearly so. I must even regard as negative the result of the experiments in which the Professor found my result "marked" or "very marked," because, in order to confirm my experiment, I must demand that the result shall be confirmative in a quite *absolute* manner. It was not so, and this is not surprising. It would have been much more surprising, had he by his method been able to confirm my results.

I know only one method for making the resection of a part of the posterior columns without damaging the rest of the spinal cord. It is the method indicated in my text-book,[1] and to which I have always returned after having examined other apparently simpler and easier methods. By it all pressure is avoided on the so-called motor tract, when we make the first incision very obliquely and very incomplete. That even a slight pressure on the (true) motor tract is very fatal in the operations on the spinal cord, is not, as Prof. Horsley believes, a new discovery of his own, but has been largely studied and set forth in my text-book, p. 278. I had given there a list of the different authors who, up to that period (1858) had been led into error by the effects of unintentional pressure on the motor elements. The resistance of these latter to pressure is less than the resistance of the sensory elements. I believe that, by this remark, I have given the key for the explanation of some paradoxical facts met with in clinical observation.

Prof. Horsley kills his dogs about the fifth day, and excites the brain with an ordinary Du-Bois Reymond's coil worked with one Daniell's cell, and in one case with two. I think that currents of so high a tension and so short duration as induction currents, are not the best nor the surest means for exciting a large parenchymatous organ like the brain. Instantaneous currents must have a very great intensity to produce a reflex movement; and the so-called "motor centres" really act by a reflex process. The intensity of the current is a cause of extensive derivations, and so we are never sure to localise exactly an induction excitation in an organ the form of which is not linear. In another paper on a recently published case of Baldi, on the brain, I shall show that all his experiments have been lost by derivation into deeper parts of the brain, and even into the roots of the fifth nerve. It is for these reasons, that in experiments on the excitable area, I do not trust induction currents, and I have always made the decisive experiments with a simple galvanic battery with a rheocord. The makes and breaks are made with the hand by means of a Morse interruptor. And indeed, in my own paper

[1] 'Physiology of the Nervous System,' p. 242. See also 'Comptes Rendus,' 1854, p. 926.

on the experiment, which forms the object of Prof. Horsley's criticism,[1] and which the Professor seems to have overlooked, I recommend for the excitation the use of 4 to 16 Leclanché cells; and only exceptionally make use of a *single* induction opening current (the hammer of the instrument being fixed) as a corroborative experiment when it gives no contraction, and as an experiment of no account, when I had an effect which could not be obtained by the battery current.

We see that Prof. Horsley has been drawn into error when he says (*l. c.*, p. 47) that my method of experimentation, as far as he knows it, exists simply in exposing and dividing the posterior column on one side. I have always made, not the *division* but the *resection* of the two posterior columns, or of one. I never excited with an inductive current rapidly interrupted by vibrations of the hammer. I made the operation on various parts of the spinal cord, always by the method indicated in my book. I made and broke the current (even the induction current) with the hand,[2] never taking it too strong. I have much insisted on this method, which I think must give to Prof. Horsley, if he will really repeat my experiments, the same result as I have obtained in my own numerous experiments.

And this result is not, as Prof. Horsley supposes that I "would have him to believe," that the so-called motor centre becomes unexcitable, which are in strict reflex relation with the muscular groups, separated from the "centre" by the resection of the posterior columns. It is quite in harmony with my results, that after the division of the posterior columns within the fourth vertebra, he should still meet with what he calls flickerings in the trapezius, and even in the face, produced by excitation of these pretended centres.

But Prof. Horsley believes that *no* excitability is lost, but that his "centre" can no longer *transmit* its excitation, because a degeneration of the pyramidal, or, as he says, motor tracts, puts a block against the peripheral extension of the innervation. Let us now examine this opinion.

[1] Pflüger's 'Archiv,' xxx. p. 257 et seq.

[2] I must add, that during the last few weeks I have found a method of using a modification of the Clarke magneto-electric machine with mechanical interruption, which can be used for experiments on the brain, without exposing us to errors produced by derivation of the current.

Can the Pyramidal Tract of the Lateral Columns be regarded as Motor?

When Prof. Horsley first published his objections against my experiments and claimed them to be produced by a lesion of the motor tract, I did not understand what he meant by this expression of "motor tract," and with its unintentional lesion. The microscopical examination of transverse sections of my spinal cords, showed no accidental degeneration. That I made this examination long before Prof. Horsley could draw my attention to it, is proved by what I said in Pflüger's 'Archiv,' 1883, p. 264, where I concluded that the operation on the posterior columns did not produce a degeneration, neither total nor partial, of the pyramidal tract. But I must add here, that only a small part of the posterior grey horns was filled with white corpuscles, the nerve fibres being intact; and there was not even in these horns any trace of descending or ascending degeneration.

But why did I not understand the meaning of Prof. Horsley? I believe I have established, in my book of 1858 (and in the previous communication published by the Natural Society of Berne and the Institute of Paris), the first lines of an experimental physiology of the spinal cord. My researches have held their ground against many contrary results, which since that time have been announced, soon to disappear. So I was accustomed to my own terminology, and I did not acknowledge the existence of a special motor tract. In Prof. Horsley's last paper I find the explanation of his meaning, and it is on this occasion that he sets forth his discovery of the influence of pressure on the motor, or more properly *kinesodic*, elements of the spinal cord we have already spoken of. He had, by his method of operating, produced a more or less extended degeneration of the undivided, but compressed, lateral tract, and he erroneously supposes that I must have done the same, and that this assumed degeneration of the pyramidal tract of the lateral columns *can*, or as he thinks *must*, cause an interruption of the transmission of movement from the brain to the limbs.

The degeneration he produces enters into the category of what the French school has hitherto called a *sclerosis*. But I

not about to combat, but I have combated for a long time this general opinion (in the case of dogs and cats), by a series of very clear and non-equivocal experiments.

My old opinion, resulting from a large number of experiments, that the lateral column does not contain fibres the section of which produces motor paralysis—an opinion which has since been fully confirmed by other experiments—may have been regarded as insufficient, as having been formed in a period (1850–58) in which no special attention has been paid to some special reflexes, which are distinguished as voluntary and unintentional, from these movements which depend upon a general and pre-reformed mechanism.

Pflüger's 'Archiv,' vol. xxx. (1883, pp. 243–248) contains a series of experiments with complete section of the pyramidal and partial section of the cerebellar tract (on one side), which prove that not only the general movements, but even the intentional ones (such as a dog giving the paw, holding the bone with both anterior legs, a cat climbing and catching objects with the limb on the operated side) are preserved, though there was not only interruption, but also a complete descending degeneration of Horsley's so-called motor tract. The degeneration, though it did not extend down to the origin of the roots of the sciatic nerve, but stopped somewhat higher, was *more complete* than that generally visible after the extirpation of the excitable area of the brain. These degenerations, and their differences in extent after injury of the brain or transverse section of the pyramidal tract of the spinal marrow, have been studied in the same animals by Prof. Löwenthal.[1]

Within the last few months these facts have been independently confirmed by Herzen and Löwenthal in a young cat, in which they had cut the pyramidal tract of the lateral column. This column showed very extensive degeneration.[2] Herzen has very well described the voluntary movements, though the cat was not kept alive long enough to allow them to be fully re-established with reference to their strength.

[1] See his communication in Pflüger's 'Archiv,' xxxi. p. 350, with the accompanying Table IV.

[2] See 'Archives de Physiologie,' April 1886, p. 20, and the plates.

have proved long ago that several forms of this so-called sclerosis do not destroy the cylinder-axis in the nerve-fibres, but are an expression of what I have called an Atelectasis of the nervous tracts.[1] Westphal's criticism of my view could not shake, but rather confirmed it, leading me to new confirmatory researches. In my paper on Atelectasis (1880), I arrived at the conclusion that, in many cases, the so-called sclerosis cannot interrupt the transmission of nervous influence—a conclusion which I applied at that period to the cases of posterior sclerosis described by Friedreich, Gowers, Pick, and others, where the sensibility was not at all, or scarcely, disturbed. Last year a new confirmation of my views arose from the discovery made in Paris, that a certain number of cases of sclerosis of the pyramidal tract presents no secondary descending degeneration. But a true block in the transmission, a transversal interruption, is always followed by descending degeneration of the pyramidal tract. Hence these cases of sclerosis do not produce a block. And this conclusion has been reached even by some neurologists, who formerly were my adversaries on the question of Atelectasis.

Degeneration of the pyramidal tracts is not produced in the experiments made according to my old method. I may regard as non-existing some experiments which I made after another method, with the "microtome" of Strauss-Durckheim,[2] in which the lateral tract was really injured.

But I can prove that this degeneration of inflammation of the pyramidal tract, even if we suppose with Prof. Horsley, and on the evidence of microscopical examination, that it has been produced in *all* my experiments on the posterior columns, *cannot be made responsible for the partial excitability I found in the so-called motor centres of the cortex.*

Prof. Horsley remarks, p. 47 of his paper, that "English neurologists, at any rate, will be interested to learn (*vide* 'Lancet,' 20th February, 1866) that Prof. Schiff is about to combat the general opinion, that the crossed lateral tract is motor." They will be still more interested to learn that I am

[1] See my paper on "Atelectasis Medullæ Spinalis" in Pflüger's 'Archiv,' xxi. p. 238, and also the criticism of Westphal in his 'Archiv.'

[2] See his 'Traité d'Anatomie comparative:' Paris, 1842, tome i. planche iv. fig. 59.

true to the principle and the method indicated in my text-book of 1885, p. 231. I can give credit even to those former experiments establishing *that a complete degeneration of the pyramidal tract, and of all the posterior half of the lateral column, does not produce in our animals loss or diminution, either in the voluntary and isolated, or in the general movements of the parts behind the section and behind the beginning of the degeneration.*

There was still one question open, and it is for this purpose that I began two years ago a new series of experiments, conducted according to my old trustworthy method. Does excitation of the excitable area of the brain, opposite to a complete degeneration of the pyramidal tract, still produce movements of the limbs of the degenerated side? Some weeks or some months after the section of the posterior part of the lateral column (pyramidal tracts) of one side, I began the experiment by exciting the brain with a battery current, and in some cases a single induction shock. The (reflex) movements in the limb, which are normally seen by stimulation of the excitable area, were present by excitation on *both* sides of the brain. The section of the pyramidal tract did not hinder or enfeeble the effect observed in the experiments of Hitzig and Fritsch. The experiments of Ferrier (with quickly interrupted induction currents) probably do not fail in these cases; but if I had excited the brain immediately after having cut the pyramidal tract, the stimulation would have remained without result. It is essential to let the traumatic irritation pass off before testing.

When in a dog or a cat, in which one pyramidal tract has been divided and has long been degenerated (from the cervical region downwards), we make a deep and extensive extirpation of the so-called "motor-centres" of the opposite side, we can before traumatic equilibrium is reached, *i. e.* in the first weeks, observe all the symptoms which have been regarded as proving the absence of intentional movements on the side opposite to the operation, and the characteristic anæsthesia is present and maintains itself indefinitely. Hence all these symptoms produced after the second operation cannot be dependent upon the existence of the pyramidal tract, in which our previous operation had caused to degenerate.

Though the full energy was still wanting, it is clear that the movements were all present.

We cannot suppose that, in these animals, the preserved *direct* pyramidal tract in the anterior columns has in some degree compensated for the destruction of the lateral crossed tract, because we know now that, at least in dogs and cats, there is nothing of the nature of a direct pyramidal tract.

The experiments of mine I have just mentioned are not decisive. At the period I made them I was using the microtome of Strauss,[1] which must be introduced by stretching, and so I produced indeed extensive lateral compressions and degenerations outside and below the divided parts. Microscopical examination of the hardened marrow[2] revealed afterwards that these errors were due to the same conditions, which Prof. Horsley wrongly supposes complicated my sections of the posterior columns. And so I produced some symptoms, which at that time I attributed to the absence of the transmission in the pyramidal tract, but which really depend upon a concomitant alteration of the posterior columns, which has been found later in the hardened parts. But even before the microscopical examination I expressed my surprise (*l. c.*, p. 243) that what I took for the effect of section of the pyramidal tracts, resembled so much the symptoms of resection of the posterior columns, and that an intentional transversal section of the pyramidal and the posterior columns made together, and simultaneously, produces no other effect than section of the posterior column alone.

So I committed at that time the error recently imputed to me by Prof. Horsley, but quite in an inverse sense; I attributed too much to the pyramidal tracts, the section of which, as we know by later and more correct experiments, produces no visible symptoms, not visible at least with our methods of research.

But even in the former vicious experiments all the movements were present; voluntary, isolated movements were not wanting, though they were modified by insensibility, and so

[1] See Pflüger's 'Archiv,' *l. c.* p. 241.

[2] See Löwenthal, 'Des Dégénérations secondaires de la Moëlle Épinière.' Thèse; Genève, 1885.

It is evident that the suppositions of Prof. Horsley, and his mode of viewing my experiments on the posterior columns, are quite erroneous. They cannot be admitted, even if it were true that I had compressed what he calls the motor tract. And they must be rejected *à fortiori*, because I had not compressed this tract.

The crossed pyramidal tract finds its nutritive centre in the neighbourhood or within the excitable area, but there is no known functional relation between this area and the lateral columns of the spinal cord.

When in 1853, after Waller's and my own experiments, I first proposed and defended the difference between the nutritive and the functional centres, there was among anatomists and physiologists much discussion and contention about this subject. Here we find another and a very striking example of this difference. This example must hold good, at least till we know something of the functions of these pyramidal tracts. It seems, after an observation of Beck, that they can be cut even in man without the loss of voluntary movement.

One of my pupils, Mr. Schopen, is now about to publish some observations of injury of the anterior part of the brain on one side accompanied by anæsthesia (for tactile excitations). In one of these cases (and in two similar I observed some years ago) the secondary degeneration was wanting in the pyramidal tracts. In two other cases there was degeneration of equal extension and intensity in the two crossed pyramidal tracts,[1] that is, on *both* sides, and notwithstanding anæsthesia and its allied symptoms were present only on the side opposite to the cerebral injury. The extirpation in these cases was not in the posterior limb of the sigmoid gyrus, but between the sulcus cruciatus and the frontal (supra-orbital) lobe. Without entering here into particulars, it is evident that such cases offer new proofs, that the functional and the nutritive disturbances are independent one of the other.

[1] The extension of the degenerations and the number of preserved nerve-fibres have been determined with the polarising apparatus. Two determinations were made for each section, one simply with crossed Nicol's prisms and one with the aid of a selenite film.

Functions of the Posterior Columns.

The preceding exposition shows, that the opinion of Prof. Horsley is not admissible, when he says that all we have hitherto attributed to the posterior columns may be due to a concomitant lesion of the lateral columns. He states, that in the cases of section of the posterior columns in which only a very slight lesion was found in the lateral, he could not verify the tactile insensibility found by me, and he did not investigate the insensibility for cold found by Herzen. That he could not verify the loss of tactile sensibility in all cases may depend either on the same causes which hindered Ferrier—to whom Prof. Horsley refers—from finding the insensibility after an injury of the excitable area of the brain, or it may depend on the method of dividing the columns. This division, as I have still to show, was imperfect in the beginning of his observations, and very slowly spread by traumatic inflammation to a perfect destruction of the column. If it is so, an examination during the first, the second, even the third day, might have given him an imperfect result.

It is probable that if Prof. Horsley had examined the dogs of this series as we examine the animals after decortication of the excitable area of the brain, he would have convinced himself that all local disturbances after these two operations are absolutely the same. I say *local* disturbances, and I should have added persistent, because the first traumatic disturbances are somewhat different. Besides, the brain is in relation with the entire half of the body (though I have hitherto been unable to produce a cerebral insensibility of the inner surface of the ear). The posterior column acts only on the parts below the operation.

Instead of this, Prof. Horsley contents himself with three methods of examination, which are very fallacious. Fallacious in the sense that their application leads into error, when they are not exactly limited and graduated as to act only on tactile sensibility and not on the other sensibilities. It is so, for instance, with reference to the "clip" Prof. Horsley mentions (*l.c.*, p. 49). It is true that very often I make use of a clip, as Goltz did before me, but I employ it rather for

demonstration than for researches. I have three pairs of clips of different strength and resistance. First, the strongest is applied successively on both sides of the body. Generally sensation is indicated on both sides. But I am not yet warranted thereby to conclude, that tactile sensibility equally exists on both sides, because the clip may act by pressure. I take a weaker one, and often a weaker still, and so I become enabled to conclude, that there exists a certain degree of contact well perceived on one side and not on the other side of the body.

The same precaution must be applied, in a still higher degree, when you touch the skin with a needle, or when you allow an object to fall on a sensitive surface, even when this object is simple water (see *l. c.*, p. 49). So it is evident that the mere enumeration of the three methods of testing used by Prof. Horsley do not warrant the exactness of his result.

These methods are not sufficient for making me believe, that Prof. Horsley has found three exceptions to a general rule, which have held good till now for many years, in very numerous experiments, without a single exception, and which has been confirmed in many species of mammals.

The ascending degeneration after resection of the posterior columns must go in some manner as far as the cortex, but I never pretended, as Prof. Horsley would have his readers believe, that it goes as far as the ganglion cells producing some visible alteration of these parts. I can add, after Löwenthal's examination of the brain of some of my dogs, that the visible degeneration does not reach beyond the pons Varolii, but it can be partly followed as far as the cerebellum.

If I have several times insisted on the fact, that I make not a section, but a *resection* of a piece of the posterior columns, I do not imply that this difference is of any importance for the result, but the difference is capital for the method. We can never make a complete section, and be sure of sparing the neighbouring parts; nay, we may be certain of damaging them.

How Professor Horsley's Results were obtained.

If it is allowable to put forth any such supposition, I think that these results are due in part to the method of operating, and partly to the mode of the excitation. Prof. Horsley makes (*l. c.*, p. 48) in a posterior column a small punctured incision, with a sharp-pointed tenotomy knife. It can scarcely be supposed that there exists a form of tenotomy knife which, with a simple puncture, divides all the posterior column, and nothing but this column. And though Horsley's figures generally show the lesion to be very exactly limited to the column, after their inspection I must assume that he made a puncture which did divide only a part, a stratum of the column, and that the rest was compressed and did disappear successively during the five days the animal was living. So a large part of the column found damaged at death may have been destroyed only three, or even two days previously. My experiments[1] have proved that one or two days after the resection of the posterior columns the brain remains excitable. In a more recent experiment, which will be communicated by Schopen, it remained still excitable four days after the resection of one posterior column. It must be partially excitable when a part of the posterior column has been divided only for two or three days.[2] So the imperfect movements perceived by Horsley after his excitation of the brain can be understood.

So we can understand how the fissure of the wound contained débris of nerve-fibres which are not present in a sharp, well-cut wound. The sides of the wound-cavity, says Prof. Horsley, "although so extremely small, showed clearly that there was some degree of tension in it." This can be understood to mean, that the walls of the fissure showed some irregularity. This must be so, even in the cases when an incomplete fissure increases by parietal inflammation. Had Prof. Horsley kept his dogs some days more, his results probably would have been more complete.

I have already spoken of the method of excitation. The

[1] See Pflüger's 'Archiv,' vol. xxx. p. 260.

[2] See the experiment of Herzen and Löwenthal on a cat. 'Archives de Physiologie,' April 1886.

use of induction currents has produced so many errors in the physiology of the brain, and more than elsewhere in the hands of English physiologists, that it is high time to give up the use of the induction coil and to return to the galvanic battery with a rheocord, which allows us to regulate the necessary variations in the strength, and in the duration of the current.

In writing the foregoing pages, it has been my aim to present the English reader with a sketch of the actual position of the question, and to give him some indications which he will seek in vain in the annual reports on the progress of physiology. I must return my best thanks to Professor Horsley for his zeal, and the interest he has shown with reference to our recent researches. I hope that this paper will engage him to undertake, on a new basis, a fresh series of experimental researches, on a subject which fully deserves the attention of English physiologists and neurologists.

A FURTHER AND FINAL CRITICISM OF PROF. SCHIFF'S EXPERIMENTAL DEMONSTRATION OF THE RELATION WHICH HE BELIEVES TO EXIST BETWEEN THE POSTERIOR COLUMNS OF THE SPINAL CORD AND THE EXCITABLE AREA OF THE CORTEX.

(*An Answer to* Prof. SCHIFF'S "*Reply*" *in this issue.*)

BY VICTOR HORSLEY, B.S., F.R.S.,

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In 'BRAIN,' April 1886, I published a paper descriptive of seven experiments I had performed, to test the value of an observation by Prof. Schiff, which was considered by that author to wholly negative the view, that the excitable area of the cortex could be considered to be motor in function.

In criticism of my paper, and in answer to my statements, Prof. Schiff wrote the "Reply" which precedes this paper. I feel therefore bound to again draw attention to my repetition of his experiments, and the more so since, in his "Reply," he has made certain definite charges against me. In my paper I made a brief criticism of Prof. Schiff's general views, in order to make clear the purport of his experiment which I repeated. I then described my work in full detail, showing that I obtained Prof. Schiff's result perfectly in some instances, but not in others.

Finally, I showed that this discrepancy was due to the fact, that Prof. Schiff's positive result occurred in consequence of an experimental accident, and that therefore his deductions from the same were unsound.

To this criticism Prof. Schiff has returned the somewhat elaborate answer which precedes this paper. In attempting to meet my criticism of his experiment, he makes some very

definite statements, of which, however unwillingly, I am compelled to take notice, and that in very full detail. Prof. Schiff's charges against me may be conveniently grouped under three headings.

I. Criticism of his views of the function of the excitable area of the cortex.

II. His experimental method.

III. English methods of experimentation on the central nervous system.

As Prof. Schiff's "Reply" is rather diffuse, I am compelled to answer him, at the risk of incompleteness, by the tedious method of collecting together all his objections under the above headings, and disposing of them one by one. And though I shall show that these objections, as such, have not really the force which Prof. Schiff's dialectical skill appears to give them, and that it is therefore unprofitable to discuss them further, I think in the interest of neurology, and especially English neurology, that it is highly advisable such statements as these of Prof. Schiff should be most thoroughly examined; for although controversy is not the best, and certainly not the most pleasant method of arriving at truth, still there can be no doubt that, by its means, facts and theories are dragged out into the light, and are thus either rescued from obscurity, to the great profit of science, or else justly condemned to oblivion.

In the establishment of a new science on the sound basis of truth, the free cosmopolitan interchange of facts and views is of the highest importance. Especially is this the case with the science of Cerebral Physiology, which, practically speaking, is not much more than a decade old; therefore, although we may differ from Prof. Schiff in our estimate of the value of the facts on which he bases his views, still, it seems to me, we are under a considerable obligation to him for having presented the question, with indefatigable energy, to us in a different light.

As neither have I the time to devote to discussion of the general ideas prevailing on the subject of cerebral physiology, nor is this the opportunity for doing so, I shall proceed to examine the objections contained in his "Reply," as

exhaustively as I possibly can in this paper, since I shall be unable, for the reasons just stated, to discuss the matter further.

I.—Prof. Schiff's objection to my criticism of his views.

Prof. Schiff says that I should not have contented myself with criticising merely his opinions as set forth by Dr. Huggard, but that I ought to have reviewed the whole of his own previous papers on the subject. Indeed, he seems to consider that I have in consequence misunderstood and misrepresented him. Whether I have misrepresented him or not, can only be determined by a comparison of what he has written in Pflüger's 'Archiv' (Band xxx.) and elsewhere, with the following excerpt from my paper, which states what I understand to embody his view.

"C. Prof. Schiff's View.—Prof. Schiff states that he has traced (physiologically) the afferent fibres (of the posterior column of the cord) directly to the excitable portion of the cortex, which he therefore regards as being the[1] sensory perceptive centre for tactile sensibility. From this sensory perceptive area he believes that fibres go down to the motor centres, which are situated he knows not where."[2]

Since this is nothing more than a comprehensive statement, in words, of the opinions expressed in his paper in Pflüger's 'Archiv,' Bd. xxx., I feel the charges of being ignorant of, and of ignoring, his previous writings to be as much without justification as they are paradoxical.

If my reading of the same was wrong, Prof. Schiff should have directly met my statement with a counter-statement, which I cannot find that he has anywhere done in his "Reply."

I shall therefore, in the absence of any correction by him, continue to consider that what I have said is a faithful exposition of his views.

Digression.

After preferring the above charge against me in the first page of his "Reply" (repeated at intervals farther on), he devotes

[1] I should perhaps have said, *part of* the sensory perceptive centre, for it is difficult to make out from his Schema, figured on p. 267, *l.c.*, that he really thinks the cortex cerebri to have any function at all.

[2] 'Brain,' April 1886, p. 43.

nearly half of his remaining space to reasserting the worth of his experiments on extirpation of the motor cortex in dogs, and to restating his deductions therefrom.

Now in my paper,[1] I particularly state that, for reasons then given, I would not discuss this experiment, and I only alluded to it, as I before explained, in order to make the object of my communication clear, viz. the criticism of Prof. Schiff's method of investigating the relation between the posterior columns of the spinal cord to the excitable area. Neither do I intend to discuss now this enormously wide subject of the representation of sensation (tactile, motor, &c.) in the excitable cortex, since I consider that the question is entirely an open one, and I am therefore unprepared either to deny or acknowledge the validity of Prof. Schiff's views on this point.

Moreover, it is not germane to the matter in dispute, viz. Prof. Schiff's method of experiment on the spinal cord.

In justice to myself, however, I cannot pass unnoticed a passage in Prof. Schiff's "Reply" by which he apparently seeks to avoid the responsibility of his whole theory relating to sensory paths and the cortex. The passage referred to is to be found on p. 28, and runs as follows:—"The ascending degeneration after reaction of the posterior columns *must go in some manner as far as the cortex*;[2] but I never pretended, as Prof. Horsley would have his readers believe, that it goes as far as the ganglion cells producing some visible alteration of these parts."

Now if the latter half of this sentence means anything at all, it says that some wonderful, and certainly never before heard of, disappearance of excitability in the ganglion corpuscles of the excitable area of the cortex follows ascending degeneration of the posterior columns. Although the said degeneration is supposed never to reach the corpuscles themselves, nevertheless it is at the same time supposed to "go in some manner as far as the cortex." It would be difficult to imagine a degeneration getting to the cortex, except as a degenerative process. But even putting the most charitable interpretation

[1] 'Brain,' April 1886, p. 46.

[2] The *italics* are mine.—V. H.

on Prof. Schiff's statement, in the interest of scientific fact I must quote again from the paper he seems to think I am not sufficiently familiar with, viz. Pflüger's 'Archiv,' Bd. xxx. On p. 259 he says, after describing the original experiment, my objection to which, in 'BRAIN,' April 1886, has caused the present controversy:—"Neu an der Sache ist, abgesehen von der durch sie gebotenen schlagenden Demonstration, nur, dass die aufsteigende Degeneration, deren obere Grenze man nicht kannte, bis zur Gehirnrinde hinansteigt. Dies soll auch bald anatomisch untersucht werden."

I need hardly ask what statement can be couched in more definite terms than this, or with more reliance upon the experiment referred to. It perfectly justified me in stating that Prof. Schiff believed the degeneration to reach (hinansteigen) the cortex; but of course I could not know that Prof. Schiff would suddenly repudiate in 1886 what he wrote in 1883. It is an instructive comment, however, on the question, that when the "anatomische Untersuchung," Prof. Schiff mentions, was made by Prof. Löwenthal (see p. 13 of his "Reply"), all the force was taken out of the above-quoted ex cathedrâ statement.

In passing to the next point, viz. Prof. Schiff's views of the representation of movement, I cannot help expressing my regret, that he should require of his readers to grant such very liberal elasticity to the meaning of his terms, as it makes discussion exceedingly difficult, and indeed well-nigh impossible.

Intimately connected, however, with the real point at issue, namely the relation of the posterior columns of the cord to the excitable area of the cortex, is the question of representation of movement in the excitable cortex, and the nature and anatomical position of the paths of nerve impulses from that portion of the brain to the spinal-cord centres. Prof. Schiff has, in previous papers, dealt with this problem at considerable length, and in his "Reply" to my criticism of his experiment on the spinal cord, has reiterated his views in plain language; so that, if I hereafter prove some of his statements to be inadequate, at least he cannot charge me with not comprehending his meaning.

I must enter here at length into a consideration of Prof. Schiff's views concerning the position of motor centres and paths, since it is by clearly keeping all the facts in view that his attack on my repetitions of his experiments can be immediately seen to be in itself perfectly contradictory.

In the first place, whereas most neurologists will admit, that Dr. Ferrier's experiments have proved that the representation of various movements is localised in certain portions of the excitable area of the cortex, Prof. Schiff places himself on exactly the opposite side, by denying that there is any motor centre in the cortex at all, and by asserting that, in his opinion, no movement whatever is represented in the cortex as such. The cerebral motor centres he apparently believes to be situated somewhere in the base of the brain. Further, Prof. Schiff believes that the fibres which make up the direct pyramidal tract, and those theoretical ones which connect the centres in the excitable cortex with his theoretical motor centres in the base of the brain, are not *motor* in function, but *kinesodic*.

With the second theoretical group of fibres we have nothing to do. Now with regard to the pyramidal tract, Prof. Schiff denies that this is a specially *motor* tract, but admits it to be *kinesodic*. Most neurologists, perhaps, would not distinguish between these terms *motor* and *kinesodic;* but in the 'Comptes Rendus,' 1854, p. 930, third line from the top, he says ". . . . . et il m'a paru qu'il fallait les distinguer des fibres sensitives par un nom nouveau, et je propose de les appeler fibres *esthésodiques* (ὁδὸς, path), et je prouverai plus tard qu'il y a, dans la même substance, des fibres très analogues pour les mouvements que j'appellerai *kinésodiques*."

Whether he calls the pyramidal tract motor or kinesodic matters nothing in the present question, for my simple contention was that it conveyed *efferent* impulses from the "motor" cortex, and consequently that a block in it would explain the result of the experiment.

At any rate, it is perfectly clear that Prof. Schiff refuses to call the pyramidal tract motor. What then, it may be asked, does he consider to be its function, and what is its relation to the excitable area of the cortex? Fortunately, he has summed

up his ideas on this subject in one passage (Pflüger's 'Archiv,' Bd. xxx., p. 249, bottom line):

"Die Hinterstränge bis ins Hirn hinein bilden die Legislative, die Türck'schen Bündel erwecken die Exekutive."

From this it is quite clear that he believes the posterior columns carry impressions into the brain, and that the pyramidal tract carries impulses from the brain which set the muscles in action. But the latter of these beliefs is exactly what I have contended to be the case; and the only difference between myself and Prof. Schiff lies in the fact, that I prefer to call the pyramidal tract *motor*, while he considers that the only term applicable under the circumstances is *kinesodic*.

Let us now follow Prof. Schiff a little further, and seek from his writings to discover what he believes to be the *functional* relation between the motor cortex and the pyramidal tract. If we simply trust to what I have just quoted from Pflüger's 'Archiv,' it would seem superfluous to do more than accept his dictum, that the pyramidal tract "erweckt die Exekutive," if he had not in most emphatic language in his "Reply" preceding this paper declared, that (p. 25) "The crossed pyramidal tract finds its nutritive centre in the neighbourhood or within the excitable area, but there is no known functional relation between this area and the lateral columns of the spinal cord," *i.e.* the pyramidal tract. Now if there is no functional relation, how comes it to "erwecken die Exekutive"? But a still more pertinent fact tending to elucidate this contradiction in Prof. Schiff's statements is one of his own experiments, upon which he based the idea of the pyramidal tract being executive to the motor area. The experiment I would refer to is described as follows (Pflüger's 'Archiv,' Bd. xxx. pp. 248, 249, commencing eight lines from bottom of p. 248):

"Einem gehörig ätherisirten Hunde wurde z. B. die *linke* vordere Hirnoberfläche und das oberste Lendenmark blossgelegt. . . . (unimportant details) . . Eine Kette von 4 Elem. kleiner Leclanché erweckte von der Gegend des Gyrus sigmoideus aus starke Zuckung entweder in beiden Extremitäten der rechten Seite, oder weiter oben nur im Hinterfuss. Es wurde auch eine Stelle bestimmt, die nur auf die vordere

Extremität und den Kopf wirkte. Jetzt wurde rasch mit dem Mikrotom der rechte Türcksche Bündel (mit seinen unvermeidlichen Begleitern) durchschnitten ohne Blutung. [The pyramidal tract was thus divided opposite the first lumbar vertebra.] *Keine Hirnreizung*[1] mit 4 Elementen erregte mehr Bewegung im rechten Hinterfuss, wohl aber in der vorder Extremität, u. s. w."

* * * * * *

(Continuation, p. 249, line 15 from bottom.)

"Durchtrennung der Bündel am obere Halsmark. *Nach mehreren Tagen*[1] Aetherisation, Blosslegung des Gyrus sigmoideus und seiner Umgebung. Vergeblich wurde sogleich und bei Nachlass der Betäubung nach einem Hirnfelde gesucht, dessen Reizung selbst mit starken (Ketten- oder Induktions-) Strömen Hitzig'sche Zuckungen gegeben hätte. Selbst Einbohren der Elektronadeln blieb erfolglos.

"Endlich wurde, um den Verdacht einer möglichen Wiederherstellung auszuschliessen, die Hirnreizung bei Hunden vorgenommen, denen schon vor Wochen der Bündel [*i.e.* the pyramidal tract] auf einer oder auf beiden Seiten durchtrennt worden. Der Erfolg war wesentlich derselbe und auch hier der Höhe der Durchschneidung entsprechend verschieden."

Now I have quoted this experiment at full length because it expresses a fact, and not a theory; a fact, too, upon which the whole of my criticism of Professor Schiff's experimental investigation of the relation between the posterior columns of the cord and the excitable area of the cortex depends. In the first part of the just-quoted experiment, Prof. Schiff demonstrates irrefragably that, if the pyramidal tract be divided, the constant result is absolute blockage in the path of the nerve-energy from the cortex to the spinal centres, that energy being evoked by stimulating the cortex. He thus *proves* that the pyramidal tract is the path of efferent impulses from the excitable area. It is therefore perfectly incomprehensible to me how he can assert, as he has done (p. 25), in his "Reply" to me, that there is no functional relation between the excitable area and the pyramidal tract. His statements of 1883 and 1886 are absolutely contradictory.

[1] The *italics* are mine.—V. H.

I prefer to believe the experimental fact of 1883, and not the controversial theory of 1886.

Further, it is extraordinary to my mind that Prof. Schiff, in his anxiety to contest the propriety of calling the pyramidal tract motor, and desiring us to adopt his expression kinesodic, should thus ignore the statements and experiments contained in his original writings, to the study of which he so frequently refers me. In his "Reply" it will be seen that he elaborates at great length the statement, that the motor disturbances (for his sake we will not call them paralyses) produced by section of the pyramidal tract in the dog are not permanent; while he wholly ignores the well-known fact, that in the dog, fibres pass down both sides of the cord from each hemisphere; the recovery of power, when only one tract in the cord is injured, being thus easily explained. Besides, we do not know what functional relation the fibres in the rest of the antero-lateral column bear to the "motor" cortex; and finally, the question whether the effects of section are permanent or not, has nothing to do with the point under discussion—viz. whether the pyramidal tract conveys efferent impulses or not.

As I must now leave this point, I trust it will be remembered that Prof. Schiff, as I have just shown, has himself demonstrated, that *damage to the pyramidal tract forms an almost absolute block to the passage of impulses from the excitable area of the cortex to the muscles.* It will be unnecessary to review at further length Prof. Schiff's views as to the situation of motor centres and paths in the brain and spinal cord, since I have quoted sufficiently from his writings to demonstrate that he has proved the pyramidal tract to be "efferent," and since he has not himself succeeded in discovering the situation of any motor centres in the brain.

II.—Prof. Schiff's objections to my criticism, and to my repetition of his experimental method.

This point, viz. the discussion of Prof. Schiff's experimental method of demonstrating the relation which he believes to exist between the posterior columns of the spinal cord and the opposite "motor" cortices respectively, is *the* source of disputation between us. I have, however, written the foregoing pages concerning Prof. Schiff's statements and theories re-

garding his view of the cerebral "motor" centres, since I consider that, in such an early and critical stage as that which cerebral physiology has arrived at, no pains should be spared to render perfectly clear the lines of thought which dictate the interpretation by others of experimental phenomena bearing on this subject.

I have said sufficient to show that Prof. Schiff believes the so-called "motor" cortex to be connected with the spinal centres by means of an efferent channel called the pyramidal tract, and further, that when this is injured, it is no longer possible to obtain movements by stimulating that "motor" cortex to which the injured pyramidal tract serves as a conductor of out-going impulses. Reference to the paper I published in 'BRAIN,' April, 1886, will show that I there demonstrated that occasionally, after section of the posterior columns of the cord, there ensued degeneration in the neighbouring pyramidal tract, close to the seat of operation. Again, that when the division of the posterior column was very exact and the wound healed at once, no degeneration occurred in the pyramidal tract. In the former series of cases, excitation of the opposite motor cortex was followed by the negative result, ascribed by Prof. Schiff to depression of function in that so-called motor area, due to the ascent of the degeneration (or degenerative influence?) process. Finally I showed that when, as in the second series of cases, there was no block in the pyramidal tract, this negative result of Prof. Schiff did not occur. From these facts Prof. Schiff seeks to dissent in two ways: first, by stating in fairly clear language (which I shall presently quote in full) that I have not obtained his result; and secondly, that my experimental method bristles with errors. These contentions I shall now rebut.

Professor Schiff's assertion, that I have not obtained his result, is introduced by him in the following words (p. 15): "Prof. Horsley, without being acquainted with our method of experimenting, believes he has repeated our experiments. His results were, in general, negative, or nearly so. I must even regard as negative the result of the experiments in which the Brown Professor found my result 'marked,' or 'very

marked,' because, in order to confirm my experiment, I must demand that the result may be confirmative in a quite *absolute* manner. It was not so, and this is not surprising. It would have been much more surprising had he by his method been able to confirm my results."

I shall content myself with meeting this very distinct charge against my experiments, of incompleteness, and indeed inadequacy, by comparing his own description of his own experiment, word for word, with my description of my repetition of the same, and I think the *absolute* and confirmatory character of my observation will be "surprising" even to Prof. Schiff.

PROFESSOR SCHIFF'S EXPERIMENT,

As described by himself (Pflüger's 'Archiv,' xxx. p. 258).

[Translation.]

"Now if in a dog or cat which has been operated upon, *i.e.* by division of both posterior columns, lower down (*i.e.* opposite 4–5 cervical nerve), and comparatively recently, and which, in spite of the residual ataxy, can move about actively, the sigmoid gyrus and adjacent cortex be exposed, and stimulated with an induction current obtained by closing the circuit of a small Leclanché battery, *no trace of movement will be obtained in either the fore or the hinder limb*, not even a twitching, although the current be very strong. *Only the facial muscles* contract; and if the strength of the current be increased, *the neck muscles* also, when the centre for the head and neck is excited; this, too, setting the hind foot in action sometimes."[1]

MY REPETITION OF HIS EXPERIMENT.

'Brain,' *April* 1886, p. 60.—[Abstract of full notes.]

Experiment V.—Dog. Adult Mongrel terrier. *Sept.* 11*th*, 1885.—Left posterior column divided (*i.e.* opp. 4th cervical nerve). 12*th*.—Dog apparently well. 13*th*.—Marked paralysis of left forepaw. Lies habitually on left side. In attempting to walk, frequently falls to left side (hemiplegia). 14*th*.—During the previous night symptoms of spinal meningitis noticed.

[1] The language in the two accounts is practically identical where placed in *italics*.

"2.20 P.M.—Both forelegs rigid, general rigidity of trunk, &c., with the other classical symptoms of meningitis. Dog anæsthetised with ether.

"2.45 P.M.—Both sigmoid gyri exposed within two minutes, and then stimulated as above described.

("I may here take this opportunity of stating, that in this series of experiments the apparatus of stimulation were an ordinary Du Bois Reymond induction coil, one Daniell cell [in one case, two cells were used] and usual platinum electrodes. The divisions indicating the distance of the secondary from the primary coil are given in centimetres; the strongest current, therefore, being at zero.)

Current.	Right Sig. Gyrus.	Left Sig. Gyrus.
10 cm.	Nil.	Nil.
8 cm.	Nil.	Nil.
6 cm.	Flickerings in left trapezius	Fibrillar twitchings in right trapezius and protrusion of limb with extension of fore-paw.
5½ cm.	Ditto.	Ditto, stronger.
Interval, *i.e.* rest of five minutes.		
5½ cm.	Ditto. no spread.	Ditto, and spread to head, and right lower limb.

"'The difference was so extremely marked as not to need further investigation.'

"In fact a superficial examination would have led one to state that the right sigmoid gyrus, as far as the centres for *the limb- and trunk-muscles respectively were concerned, was inexcitable.* As a matter of fact, however, in this, as in all other experiments, the right cortex are perfectly excitable, as evidenced *by the active movements of the facial and cervical muscles* as low as the distribution of the fourth cervical nerve. I have already described the condition of the spinal cord in this case (see page 18), in which description it will be remembered that the degeneration of the motor tract was most marked. This case will thus serve as a typical illustration of the mode in which I believe Prof. Schiff's result is always produced. Of course it was impossible in this instance that the nerve energy should reach the left limb muscles."

I leave it to the judgment of my readers whether, with the

exception of minute details, which are set forth at length in the account of my experiment, the result I obtained was not exactly the same[1] as Prof. Schiff's, according to his original account of it. I think it will be seen by the above quotation that the charge he so frequently brings against me of omitting to refer to his original articles on this subject is not only unfounded, but also actually recoils on himself.

As I have now shown that the result of our experiments are perfectly identical when the pyramidal tract was blocked, I shall now criticise in detail his experimental procedure, a criticism which I should not have thought of undertaking, had he not so frequently accused me of neglecting to acknowledge or quote (and indeed read!) the sources whence I obtained my ideas of his views and methods.

(1.) Division of the posterior columns of the cord.

Prof. Schiff says that he effects this not as I naturally concluded from his continually calling it "Trennung" and "Durchschneidung," by a simple cut, but by performing a resection of a certain length of the column. This he describes distinctly in two places, the most recent of which I give first because it now appears that he has abandoned it for the older method. The later method he gives, Pflüger's 'Archiv,' Band xxx., page 257, five lines from the top.[2]

"Mehreren Hunden und Katzen habe ich, in der obern Cervikalgegend, die beiden Hinterstränge des Rückenmarks in ihren ganzen Breite durchschnitten und dann die Schnittwunde in der Tiefenrichtung durchziehen mit der Pinzette vervollständigt"!

The older method he gives in the 'Comptes Rendus,' 1854, p. 926.[3]

"J'ai fait une incision qui divisait transversalement la plus

[1] Note particularly the sentences in *italics* (mine).—V. H.

[2] *Translation.*—"In several dogs and cats I have cut through both posterior columns in the upper part of the cervical region of the spinal cord, and then deepened the wound by dragging out portions with forceps"!

[3] "I made an incision which divided transversely the major part of the posterior columns near the caudal extremity of my wound, then seizing the cut portion with forceps, I contrived, by employing gentle but steady traction forwards and upwards, to isolate not only the part divided, but also the whole mass of the posterior columns longitudinally for a distance of several centimetres."!

grande partie des cordons postérieurs près du bout caudal de la plaie, puis saisissant, avec une pince la partie coupée, j'ai pu à l'aide d'une traction douce, mais continue, en haut et en avant, isoler non seulement la partie coupée, mais toute la circonférence des cordons postérieurs dans la longueur de plusieurs centimètres"!

We learn from Prof. Schiff's interesting "Reply," p. 299, that he has abandoned his cutting (plus dragging!) operation for the combined cutting and excavating method I have just quoted from 'Comptes Rendus.' I cannot admit that either is an exact method of producing experimental lesions in the spinal cord; in fact, I cannot conceive of any operative procedure more likely to be followed by traumatic complications. I cannot anywhere find in Prof. Schiff's writing a distinct proof by microscopical investigation of the harmlessness of this method; indeed, he only says in 1854 that naked-eye inspection of the cord, after hardening in alcohol, revealed no damage to the surrounding parts, and therefore further discussion of this method appears to me waste of time. Although Prof. Schiff appears to have read my paper, he asserts that I divide the column by means of a puncture only; whereas, had he quoted me correctly, he would have said a puncturing incision. However, since I publish microscopical appearances at the point of section of each of the cords I experimented upon, I need not dwell further on my method, since any one can form their own judgment on this matter by referring to my drawings.

Prof. Schiff makes the somewhat indefinite suggestion that I damaged the pyramidal tract by pressure during the operation. I have not the least idea how such a thing could occur; but if we imagine it to happen, I submit that his method is equally open to the same error. I think I had better close the consideration of this part of the matter by stating that I gather Prof. Schiff's observations to have reference to my belief that the degenerative block in the pyramidal tract is partly brought about by tension in the small wound cavity in the cord, together with, of course, as I stated in my former paper, interference with its blood supply, &c.

However this may be, it is obvious from my specimens that when the operation of division of the posterior column was accomplished neatly and without complication, that there was no block in the pyramidal tract, and that, as a necessary consequence, Prof. Schiff's result was not obtained. The reader will no doubt now fully understand why I quoted Schiff's own experiment on p. 5, showing that damage to the pyramidal tract produced the very phenomenon which, in the same paper, he attributes to the ascent of a hypothetical degenerative influence to motor cortex from the divided posterior column.

Since I am compelled to be a little discursive in order to refute Prof. Schiff's charge, I must now rebut his statement, that the block I observed in the pyramidal tract, in those experiments in which his result was obtained, was not a true block, he suggesting that I said it was a sclerosis. Having thus fathered upon my description a misleading name, he tells us, p. 302, that he has previously demonstrated that sclerosis in man does not affect the axis-cylinders. All this byplay of Prof. Schiff has nothing to do with the point under discussion, because in my paper, which it is clear he cannot have read, to say the least of it, carefully, I state in full detail that the block is brought about by degenerative changes in the axis-cylinders themselves.

Prof. Schiff repeatedly charges me in his "Reply" with ignoring, or being ignorant of, his original statements. I have already shown that he must himself have forgotten his own writings, and I must now add that he appears ignorant of, or to ignore, mine also.

(2.) Clinical methods of examining the disturbances of tactile sensibility after the division of the posterior column.

Prof. Schiff takes exception to the means I adopted for testing tactile sensibility in my experiments. As the first, namely the clip, is a method which he himself devised, and upon which stress is laid not only in Dr. Huggard's paper, but also in his original article, I cannot conceive why Prof. Schiff finds fault with me for adopting it. Further, the second, viz. gentle touching with a point, is that which is universally employed all over the world for the purpose; and as to the third, Prof. Schiff refers to it in a comprehensive

sentence of intended depreciation, viz.—"So it is evident that the mere enumeration of the three methods applied by Prof. Horsley do not warrant the exactness of his result." It is obvious that I cannot discuss such criticism as this.

(3.) How Prof. Horsley's results were obtained.

Prof. Schiff concludes his review of the experimental methods I adopted by a paragraph headed as above. Some points in his conclusion I have already shown to erroneously arise from an imperfect reading of my paper. I have now to finish all I ever intend to say on this subject, by rebutting the few facts finally alluded to by Prof. Schiff.

In the first place, although he admits that my lesions were generally very perfect, he nevertheless attempts to diminish their scientific value by pointing out that in one or two instances there was a minute fragment of the column left undivided. The general frailty of Prof. Schiff's position is here demonstrated by his drawing attention to this fact, since, as will be seen by reference to my paper, his result was actually well marked in these very experiments, while with one exception it was absent in those experiments in which the column was absolutely divided. I note further an expression in connection with this point, which Prof. Schiff uses—an expression which may mean a good deal, viz. "this must be so even in the cases when an incomplete slit increases by parietal inflammation." If Prof. Schiff really means inflammation, it is perfectly clear to my mind, that in his experiments strict antiseptic precautions are not successfully carried out. As in all my own researches, I reject all experiments in which healing does not take place by so-called immediate union, I searched Prof. Schiff's original articles, to find whether he followed the same course. It has not been in my power to consult the Italian papers he refers to, but not in the 'Comptes Rendus,' nor in Pflüger's 'Archiv,' where his original and most important papers were first published, nor in his 'Lessons on the Physiology of the Nervous System,' have I been able to discover reference to this experimental detail, which, is in my mind, of absolutely primary importance in planning experiments of this kind. I have dwelt on this point not so much with the object of discussing its importance as of drawing attention again to the fact that, in

those of my experiments, which I considered failures, owing to the breaking down of the antiseptic treatment of the wound, Prof. Schiff's result was easily obtained; whereas, in the instances in which immediate union took place, and in which there was, of course, no "parietal inflammation" of the wound-cavity in the spinal cord, his result was either absolutely wanting or very slightly marked.

It is obvious to my mind, as I pointed out in my first paper, that such a fact as this, taken in conjunction with the further fact of there being an anatomical block in the pyramidal tract, makes my former conclusion, which I have in this paper demonstated to be confirmed by one of Prof. Schiff's own experiments, perfectly certain; viz. that his experimental method does not prove that section of the posterior columns of the cord causes a loss of function in the opposite so-called motor cortex.

In the second place, the mode of excitation of the excitable area of the cortex is a point upon which Prof. Schiff holds very definite views, and these he has given us in clear language: thus, he says, "I have already spoken of the method of irritation. The use of the induction current has produced so many errors in the physiology of the brain, and more than elsewhere under the hand of English physiologists, that at length it is time to give up the use of the induction coil—to return to the battery with rheocord which allows us to vary as we need the strength, the increase, and duration of the current. Elsewhere, page 8, he says: "The intensity of the current is a cause of active derivations,[1] and so we are never sure to localise well an induction irritation in an organ the form of which is not linear." He further goes on to suggest that, owing to the escape of current, motor centres, which he imagines to exist at the base of the brain, but which he has never been able to find, are irritated thereby. It is difficult to understand why Prof. Schiff wrote the first sentence I have quoted on this subject, because, apart from its somewhat curt criticism of English neurological experimentalists, any one unacquainted with the facts might easily imagine that it was not possible to vary the strength, the increase, and the duration of an induction

[1] Professor Schiff here clearly means escape of current.

current, which of course is absurd. As to the old objection that errors arise from escape of current, I think really, after the many years that this has been before the scientific world, it is hardly worth while to take further notice of it than to re-quote the most obvious confirmation of the value of the method, a confirmation which receives, by the way, additional strength from its being endorsed by Prof. Schiff's own late assistant, Prof. Löwenthal, in the paper published by him and Prof. Herzen, in the 'Archives of Physiology.' This confirmation of the accuracy of the method is simply as follows. If on irritating some given spot of the excitable area the movement produced thereby be recorded, it will be found that shifting the electrodes only of 1 or 2 mm. from that spot, a different movement will be elicited. Finally, that if an excessively powerful current be employed, the result will be summation of these two movements. It is therefore quite beside the mark to suggest that the induction current is unable to differentiate the relations of the function of one spot in the brain as compared with that of another. Prof. Schiff tells us that he uses a constant current. This has long since been abandoned by English workers in this department of science, on account of its electrolytic action on the surface of the brain.

I cannot help feeling, after all, that the discussion of this well-worn subject is quite superfluous, for I have shown that I obtained Prof. Schiff's result just as distinctly as he did himself, so that the difference between the constant and induced currents has no bearing whatever on the present question. I, in fact, have only touched upon it because Prof. Schiff objected to my employment of the latter form of irritation in the repetition of his experiments.

In conclusion, I cannot help expressing a certain amount of disappointment that Prof. Schiff, in taking exception to my repetition of his experiments, should have omitted to furnish the judicial reader with the full details of his operative procedure, with drawings of microscopic sections of the cord operated upon, &c., for I can find nothing more definite even in his original paper than "Mehreren Hunden und Katzen," &c., giving scanty details of only two of these, and finally winding up with "Die Autopsie der beiden Wunden des

cervical Markes ist noch nicht gemacht." I submit, with all due respect, that to draw conclusions from experimental work of this kind is likely to be prejudicial to the progress of the physiology of the nervous system; and that I do not feel this opinion to be in any way modified by what Prof. Schiff says in his present "Reply," for he still does not furnish us with any of the details I have referred to; while I think I have said enough to justify my previous criticism. The said criticism amounting to this, that Prof. Schiff's experiment of dividing the posterior column of the cord, and subsequently discovering that on the fifth day after he could elicit no movement of the limbs on the same side by stimulating the opposite motor area, does not (owing to numerous inherent fallacies) *prove* the posterior columns of the cord to have a direct relation to that motor cortex. Further, that his result is only to be obtained when (as he himself has unwittingly demonstrated) there is damage to the pyramidal tract.

PARALYSIS OF THE MOVEMENT OF CONVERGENCE OF THE EYES.

BY H. PARINAUD, M.D. (PARIS).

(*Translated from the French MS. by* HENRY JULER, F.R.C.S.)

THE paralyses of the muscles of the eye,[1] as of those of other parts of the body, arise either from central or peripheral causes. Until the last few years this fact appears to have been ignored, for the descriptions are devoted almost exclusively to the paralyses of the third pair of nerves, and the symptomatology is exactly deduced from the distribution of these nerves and from the action of each muscle. Several works, however, have been published on this question, notably those on conjugate paralysis, paralysis of the sixth pair, and Hutchinson's ophthalmoplegia.

I may here be permitted to remark that in several publications, of which the first appeared in 1877, I have endeavoured to establish the distinction between peripheral and central paralyses. Hitherto these subjects have not seriously occupied the attention of oculists, but at the present time, thanks to to the efforts of Prof. Mauthner, this gap in the literature of ophthalmology is becoming filled up.

In my paper on "Paralysis of Associated Movements,"[2] I have insisted upon the necessity of searching for *clinical types* which shall enable us to understand the significance of those partial and complex forms which are most common amongst central paralysis.

Associated paralyses, *i.e.* those referring to similar binocular movements, are central paralyses *par excellence*. One of these is well known as conjugate paralysis of the sixth pair, thanks

[1] The cases on which this paper was written were observed, either in Dr. Parinaud's ophthalmic clinique or in the Salpêtrière, where the author fills the post of Ophthalmologist to the Neurological Department of Prof. Charcot.—ED.

[2] 'Archives de Neurologie,' March, 1883.

to the clinical observations of Prevost, Foville and Priestley Smith; to a previous case with autopsy by Féréol; to the experimental researches of Duval, Laborde and Graux; and to a memoir by Landouzy, who enlarges the subject by examining the relations existing between the hemispheres and the bulbo-pontal nuclei.

Conjugate paralysis from lesion of the nucleus of the sixth pair of nerves is not the only associated paralysis that has been observed. This is easily conceived, inasmuch as the other binocular movements in virtue of their peculiar anatomical disposition are equally associated.

Amongst other forms of associated paralysis that I have studied, I have signalised the *Paralysis of Convergence*, of which I desire to delineate the characteristic features.

It is hardly necessary to remark that this central paralysis of convergence is distinct from the insufficiency of the internal recti muscles, with which we are familiar in connection with myopia, with peculiar arrangement of the cranial bones, and from a congenital muscular insufficiency.

The deficient converging power observed in exophthalmic goitre[1] by Möbius has always seemed to me to be in relation with the ocular protrusion. In certain cases it is true this symptom may appear before the latter is very manifest. But, though the disease be primarily of nervous origin, the loss of convergence in Basedow's disease must be referred to a mechanical cause, as Möbius suggests, and must not be taken for a paralysis strictly so called.

I may call attention here to one form of insufficiency of the internal recti which is in some sense intermediate between insufficiency from ocular and that from cerebral causes. It may be called *Insufficiency from over-fatigue*, from excessive cerebral, as well as muscular, exhaustion.

In July last year three young girls were brought to me from the Ecole Normale Supérieure. They all presented symptoms of muscular asthenopia, and all the characters of pronounced insufficiency of the internal recti muscles. None of the patients were myopic. The visual trouble at first developed towards the end of the scholastic year, and after

[1] 'Centralblatt für Nervenheilkunde,' 1886.

the most assiduous labour. The use of prisms afforded some relief and enabled them to continue their studies. After the rest of the vacation, these symptoms all disappeared, and the use of the spectacles was discontinued.

There are two forms of paralysis of convergence arising from central causes. The one may be called *essential* and the other *combined*; combined, because it coincides with paralysis of elevation and depression.

A.—Essential Paralysis of Convergence.

The vision of a near object is accompanied by three distinct muscular acts; The *convergence* of the eyes towards the object looked at, the effort of *accommodation*, and the *contraction of the pupil*. These three acts are concerned in the typical forms of essential paralysis of convergence.

(*a.*) Paralysis of the movement of convergence is indicated by two principal symptoms: the objectively appreciable defect of convergence and a peculiar diplopia.

In order to recognise the defect of convergence, we proceed in the same manner as for insufficiency of the internal recti. If the patient be told to fix the finger as it rapidly approaches his face, the eyes remain immovable; if the movement be performed, it is incomplete, the patient cannot continue to look at the object with both eyes, but one of the eyes turns outwards and he squints. Lastly, in making him fix a near object alternately with either eye, the eye which has been momentarily excluded from vision executes a movement of readjustment, in order to fix the object. These are the objective symptoms of insufficiency of the internal recti.

On the other hand, in the lateral movements the internal recti contract normally. In a word, the innervation of the internal recti is only concerned in the movement of convergence; it remains normal for adduction in displacements where the visual axes are parallel. The *diplopia* appears in the median plane at a variable distance from the subject; *it is crossed, and there is moderate separation of the images which persist without notable modification in all directions of looking.* The separation is not sensibly increased when the candle is moved laterally, sometimes it diminishes, a character which

distinguishes this diplopia from that of all ordinary paralyses of the internal recti. The images not unfrequently become fused at four or five metres from the patient, a distance at which but little convergence is exercised, but cases do occur in which the diplopia persists at all ranges; this may be explained either by the degree of paralysis, or by the individual differences in the static condition of the eyes. There are subjects in whom, during absolute repose, the globes have such a tendency to diverge, that the maintenance of parallelism necessitates the intervention of converging force.

If the patient does not complain of diplopia when a coloured glass is employed, a prism, placed base upwards, before one of the eyes will generally cause the double vision to appear, and so allow its characters to be studied.

(*b.*) Accommodation is involved in this form of paralysis in various ways. In certain cases there is absolute paralysis in both eyes; in others there is more or less notable reduction of its amplitude: whilst in a third class the accommodation is normal.

Accommodation remains defective in monocular as well as in binocular vision. It cannot be admitted that it is indirectly associated in virtue of the functional association of accommodation and convergence. This explanation, more or less applicable in certain cases where the paralysis is recent, does not account for those in which the paralysis of accommodation is absolute and may persist indefinitely.

The double paralysis of accommodation is not accompanied by mydriasis from paralysis of the sphincter, which distinguishes it from certain facts of double paralysis belonging to ophthalmoplegia interna and derived from another process.

We may further distinguish paralysis of accommodation connected with paralysis of convergence from paralysis arising from reflex causes, or from those occurring after diphtheria in which there is no paralysis of the iris. In one case of double paralysis of accommodation, after diphtheria, which I recently studied from this standpoint, convergence and pupillary reflex were normal.

(*c.*) If in this form of paralysis there be no paralytic mydriasis, the pupillary reflexes are nevertheless modified, and

mostly in a very characteristic manner. *The reflex is abolished for convergence and retained for light*, thus constituting a modification exactly inverse to that pointed out by Argyll Robertson as occurring in tabes. The reflex is equally defective when the patient is made to fix a near object with one eye only. In certain cases the pupil, though not dilated, will not react to any excitation.

Paralysis of Divergence.

The association of ocular movements for fixation at different distances can be modified in another manner. Eyes may converge for short distances, but yet be unable either to bring their visual axes back to the parallel directions, or even to fix an object at the distance of one or two metres. This I have called *paralysis of divergence.*

Paralysis of divergence can hardly be recognised except by the character of the diplopia. This, in the median plane, only appears at a certain distance, *it is homonymous, with slight separation of the images, and persists without notable modification in all directions of looking.*

The paralysis of divergence is not incompatible with defective convergence. In some cases the visual axes are found to cross at a certain distance, whilst the amplitude of convergence is nil. Beyond this point the diplopia is homonymous; within it the diplopia is crossed.

Amongst other symptoms accompanying paralysis of convergence must be mentioned the difficulty of estimating distances and vertigo. The first of these is sufficiently accounted for by the paralysis of convergence and accommodation. This symptom is most marked when the visual trouble is rapidly developed. The vertigo is partly explained by the false notion as to the position of the object, and by the diplopia, but this is not its sole cause. *The majority of patients in whom this paralysis exists suffer from vertigo independently of their ocular troubles.* In some there is vertigo with a sense of translation in space, similar to that observed in Ménière's disease. It is, moreover, remarkable that aural troubles are sometimes found to be co-existent.

The essential paralysis of convergence consists, then, in the lesion of that function in virtue of which the point of convergence of the visual axes is displaced in fixation at different distances, a function which may be disturbed in two ways, by lesion of the forces of convergence, and of the forces of divergence.

As to the seat of the lesion which concerns this function, we are not able actually to arrive at a precise determination. There are some reasons for believing it to be situated in the cerebellum.

I would mention in the first place the condition of vertigo which I have found to exist in several patients, and which is independent of visual troubles. It is not necessary to recapitulate the rôle which the cerebellum is supposed to play in cases of this kind, and in the vertigo with sensation of translation of the Ménière type. Recent works render almost certain the existence of a special nerve, called *nerve of space* ("nerf de l'espace"), connected with the auditory nerve, the lesion of which produces the vertigo of Ménière. But it is not only peripheral lesions of the nerve that give rise to this; those of the cerebellum, to which it leads, can produce exactly similar vertiginous symptoms to those of the aural disease.

We form our notions of space not only by the sense of hearing, but also by that of sight. It is, however, especially by the movements of convergence of the eyes that we obtain our notion of the third dimension to which that of space is essentially allied. It is probable that this notion of space has a functional centre, whether perceived by the eye or the ear. If, in reference to hearing, there are reasons for placing this in the cerebellum, these facts are in favour of a centre in the same situation for the co-ordination of the movements of convergence.

Lastly, I will mention that the experiments of Duval and Laborde have clearly established the influence of the cerebellum upon the co-ordination of the movements of the eyes; and, a fact of importance from our present standpoint, this influence appears to bear exclusively upon the relations of the ocular axes to one another. Whilst lesions of the bulb and of the pons constantly produce conjugate deviations of

the eyes, those of the cerebellum, and particularly of the vermis inferior, produces what these experimenters call *dissociated deviations*, in which the axes of the two eyes, instead of being displaced parallel to each other, are modified in their relations, so as to produce strabismus.

At the moment of sending this memoir to the press, I observed with M. Charcot a new case of paralysis of convergence, which has more strongly fixed our attention upon the relation of this paralysis to the vertigo of Ménière's disease (Obs. IV.).

The patient, in whom the visual trouble declared itself eight months ago, in what he terms an attack of congestion, had suffered from vertigo with diminished acuteness of hearing for fifteen years. The attacks, according to MM. Charcot and Gellé, who examined the state of hearing, presented the true characters of aural vertigo, and possibly the supposed congestion which marked the outset of the diplopia was nothing more than violent vertigo.

In the presence of such a case, the question arises whether a lesion of the ear could not give rise to visual troubles by reflex influence. We must always remember that this paralysis may exist for months, years, or even indefinitely, which is hardly the case with reflex paralyses; and besides that, it may exist without these violent attacks of vertigo, and without lesion of the ear. I am more disposed to believe that the troubles of hearing and of vision arise from the same central lesion.

I am hardly competent to solve this difficult question. I will only mention that Jackson has indicated the existence of abnormal movements of the eyes during aural vertigo, and that he attributes to them a certain rôle in the mode or origin of the access.

M. Charcot, on the other hand, has observed in patients the existence of diplopia during the access. Lastly, in some cases we may see ordinary paralysis persisting in certain muscles. One patient at the Salpêtrière presented at the same time as the paralysis of convergence, an absolute paralysis of the left external rectus muscle.

I must add that many patients suffering from typical aural vertigo present no symptom of loss of convergence.

OBSERVATION I.—*Paralysis of Convergence and of Accommodation Abolition of Pupillary Reflex and of Convergence.*

M. Vern—, æt. 30, mechanic.

Power of convergence, nil.

On attempting to converge, the left eye deviates freely outwards. Adduction normal during lateral movements of the eyes.

Crossed diplopia in the median plane for all distances. Beyond 3 mètres, separation of images diminishes; it does not increase lateral directions of regard.

M = −1 D in each eye. Double paralysis of accommodation. With + 2·5 D. reads No. 1 at 30 centimetres.

Pupils equal, of normal dimensions. No reflex for convergence. Active to light. Nor does the pupil react when he fixes with a single eye.

No lesion of fundus.

Appreciates distances badly, a circumstance which he much laments, as he was formerly an excellent shot.

Visual trouble commenced with diplopia three weeks ago. Headache five years. Vertigo three months. Erythropsia.

OBSERVATION II.—*Paralysis of Convergence and of Accommodation. Absence of Pupillary Reflex.*

Madame Bodd—, æt. 52.

Power of convergence, nil.

Contraction of internal recti normal in the lateral directions of regard.

Crossed diplopia upon the median line for all distances. When the candle is displaced laterally, the separation of images instead of being increased is diminished. Diplopia persists in all directions of regard.

H. = + 1·5 D. Double paralysis of accommodation. With + 5·5 D. reads No. 1 at 30 centimetres.

Pupils equal, freely dilated.

Both reflexes abolished.

Fundi normal.

Vertigo frequently since the age of 2. Patient always feels as if she were intoxicated.

Persistent pains in the occipital and left temporal regions. Knee-jerk normal. No ataxy, no syphilis.

OBSERVATION III.—*Paralysis of Convergence and of Accommodation. Abolition of the Reflex of Convergence.*

Madame Mita—, æt. 54.

Fixation of a near object gives rise to no change in the pupils.

Patient does not complain of diplopia. This can be produced by the simultaneous use of a red glass and a prism base upwards. At 4 metres the images are upon the same vertical line. Nearer to the patient there is crossed diplopia; the separation is not increased by lateral displacements of the candle. Double paralysis of accommodation H = + 2 D. With + 6 D. pp. at 30 cm. reads No. 1.

Pupils equal, normal in size.

Reflex retained for light, abolished for convergence.

Onset undetermined, vertigo for a long time. When the patient goes out in the bright sunlight she is seized with vertigo and vomiting. Sense of numbness on the left side.

OBSERVATION IV.—*Paralysis of Convergence and of Accommodation. Abolition of Pupil-reflex in Convergence.*

Mr. Ball—, æt. 42.

Movement of convergence, nil. The lateral movements have their normal amplitude, but are accompanied by slight nystagmus. Crossed diplopia for all distances and in all directions of regard. No notable modifications of separation in the right and left directions of regard.

Pupil-reflex reserved for light, defective when convergence is attempted or when a near object is fixed with a single eye.

Paralysis of accommodation in each eye. M = −1·25 D. V = $\frac{5}{10}$. With 3 D. pp. at 30 cm. reads No. 1 Snellen.

No lesion of fundus.

Onset of visual trouble in October 1885.

After an attack, without complete loss of consciousness, the patient suffered for fifteen days from visual trouble which prevented him counting his fingers; since he has improved, he has remarked that he sees double.

For fifteen years the patient has had vertigo with slight deafness, in which MM. Charcot and Gellé have recognised the characters of aural vertigo.

OBSERVATION V.—*Paralysis of Divergence.*

Madame Lep—, æt. 31.

Neither strabismus nor trouble in ocular movements are objectively appreciable. *Homonymous diplopia* becomes evident at 40 cm. from the patient and increased beyond that distance. The separation of the images remains moderate; instead of becoming increased when the candle is moved to the right or left, it tends to diminish. The diplopia persists in all directions of regard, in the

upper as well as the lower half of the visual field. No inclination of images.

The pupils react to light and to convergence, but feebly.

Sensibility of retina normal. No ophthalmoscopic lesions.

Sudden onset of visual trouble on Jan. 1, 1884, without loss of consciousness, without other accident. No vertigo. Habitually suffers from frontal headache. No ataxy nor syphilis.

OBSERVATION VI.—*Paralysis of Convergence and Divergence.*

Madame Dup—, aged 62.

All other movements normal.

Crossed diplopia on the median plane, for a short distance.

At 1.50 metre the images become fused. At 3 metres diplopia becomes homonymous.

Diplopia persists in all directions, without any notable modification for the same distance.

Pupils normal in size, and reaction.

Sudden onset of visual trouble by diplopia without any other symptom. No vertigo.

B. COMBINED PARALYSIS OF CONVERGENCE.

In this form of paralysis, the defect of convergence is accompanied by paralysis of elevation, and of depression in both eyes, with retention of lateral movements.

This paralysis appears to be very rare in its typical and complete form. I have seen one case where the visual trouble, which had come on suddenly in a polyuric patient, was clearly characterised. The following is a résumé of the observation, reported more fully in my memoir on *Associated Paralyses.*

OBSERVATION VII.—*Paralysis of Convergence with Paralysis of Elevation and Depression in both Eyes. Retention of Lateral Movements.*

M. Sey—, æt. 67.

Movements of elevation and depression almost nil in both eyes.

Complete paralysis of convergence.

The internal recti contract normally for adduction in lateral movements.

The diplopia is crossed or homonymous, according to the distance of the candle. The image of the right eye always remains rather the lower.

The elevators of the lids are intact.

No paralysis of iris; there is, on the contrary, slight myosis with abolition of reflexes.

The amplitude of accommodation is almost nil, but, knowing the patient's age, one cannot ascertain whether this be paralysed.

The paralysis appeared suddenly on the night of Feb. 8, 1881. Since that date there has been a tendency to fall to the left. Polyuria recurring in crisis, with heaviness of the head, from which the patient suffered before the development of the ocular trouble. Pulse 52.

In an observation by Wernicke, quoted by Nothnagel, we find the same type of paralysis. The two eyes are unable to move either upwards or downwards, whilst lateral mobility to the right or left remains perfectly normal. But the condition of the convergence is not pointed out.

This form of paralysis may be incomplete. With paralysis of convergence we may have only paralysis of elevation (Observation V. in my memoir), or paralysis of depression (Observation by Priestley Smith[1]).

These symptoms correspond exactly to those of paralysis by lesion of the nuclei of the 3rd pair, as we take it to be along with integrity of the nuclei of the 6th pair, which are well known to innervate the external rectus of the same side and the internal rectus of the opposite side, but only for the lateral movements.

The extension of the paralysis to both eyes may be explained by the alteration of the two nuclei which are sufficiently near to each other to be simultaneously attacked by the same lesion. Moreover it is not impossible that the lesion of a single nucleus may produce bilateral results. This hypothesis has already been advanced to explain certain cases of labio glosso-pharyngeal paralysis (oral communication by M. Charcot[2]).

Finally, it is not impossible that the lesion, instead of directly attacking the bulbo-pontal nuclei, is situated in a

[1] 'Ophth. Hosp. Reports,' 1876.

[2] I do not know of any very conclusive observations on paralysis of the 3rd pair relating to one eye only in which *the movements of adduction of the internal rectus are conserved* in the lateral directions of looking. Therefore in considering typical nuclear paralysis we must suppose each nucleus to innervate one eye only. The following remarks bear upon a case in which this form of paralysis was well characterised, but the affection appeared to be congenital:

neighbouring centre, which acts immediately upon them, and it appears that this centre may be the tubercula quadrigemina. This appears to be the result of Wernicke's observation (paralysis of elevation in each eye, with conservation of lateral movements), where the autopsy proved a lesion of the *right corpora quadrigemina*, and of another by Hénoch[1] in which the paralysis of elevation in each eye was connected with a well-defined tubercular lesion of the *left posterior quadrigeminal body*.

It has been long known that connections exist between the oculo-motor centres and the quadrigeminal bodies. In this direction we shall bear in mind the recent experiences of Darkchewitch,[2] who confirms and delineates the existence of these connections.

Observation VIII.—*Paralysis of the 3rd pair, with Conservation of the Movement of Adduction, probably Congenital.*

Marie Bauv—, æt. 12½.

Left Eye.—Movements normal in monocular and binocular vision.

Right Eye.—In monocular exploring, the movements of elevation and depression almost nil. Adduction and abduction normal. Action of inferior oblique appears to be completely defective. Action of superior oblique, deprived of its antagonist by the movements of torsion, is, on the contrary, exaggerated. If the patient attempt to look downwards and outwards the eye describes a downward movement, and further undergoes a very marked degree of torsion, similar to the hands of a watch.

In exploring binocular vision the same results are found, and further that the movement of convergence, which is normal in the left is absent in the right eye; it is true this eye is highly amblyopic, only counts fingers.

No lesion of the fundus.

The pupil is slightly dilated, but there is no paralysis of the sphincter. The reflex is absent for convergence, it is feeble for light. The elevator of the upper lid is not affected. The affection appears to be congenital; the child has always squinted freely; she had convulsions at the age of 26 months.

[1] 'Berlin. Klin. Wochensch.,' 1864, No. 13.

[2] 'Neurolog. Centralblatt,' 1885.

NOTE ON TWO NEWLY DESCRIBED TRACTS IN THE SPINAL CORD.

BY CHARLES S. SHERRINGTON, M.B.

It is now twelve years since Woroschiloff published, among other experiments conducted under Ludwig's supervision, some which showed that in the lower dorsal region of the spinal cord of the rabbit, after both posterior columns and the posterior halves of both lateral columns, and the whole of the grey matter lying behind the central canal had been cut through, sensory impulses arising in the nerves of the hinder limbs still passed forward to affect motor mechanisms in the cervical spinal cord and brain. The tract or tracts thus transmitting sensory impulses in the anterior (ventral) half of the spinal cord remained an anatomical mystery until much more recently. The direct cerebellar tract, if that occupy in the rabbit a position corresponding to its position in man, it could not be, for, in that case, by Woroschiloff's experiment, it must have been completely cut through.

Passing over an observation of Gowers in 1877, which tended to extend Woroschiloff's result to injuries of the human cord, and, passing over scattered evidence by Westphal of the existence of tracts capable of independent degeneration in the ventral portion of the antero-lateral column, especially of the cervical region of the cord, Gowers' notice of an ascending antero-lateral tract in his 'Diseases of the Spinal Cord,' is the first anatomical confirmation of Woroschiloff's observation. Westphal undoubtedly figured at least once a degeneration of the tract elucidated by Gowers, but his material did not allow him to delimit, nor could he suggest the function of fibres degenerated. Kahler and Pick, Strümpell and Leyden, also figured portions of the same tract, but had no means of gauging its real extent or its significance.

In 1880, however, Gowers figured a preparation taken from the dorsal region of a spinal cord in a case in which the lower extremity of the cord had been crushed. The preparation was figured to show that besides the ascending degeneration in the posterior columns, an area of ascending degeneration existed in each lateral column just in front of the region of the crossed pyramidal tract. This ascending lateral degeneration gave in cross section an area shaped like an inverted comma, the tail of the comma occupying the periphery of the antero-lateral column in front of the direct cerebellar tract, the head occupying part of Flechsig's "mixed area of the lateral column." In this case the injury to the spinal cord had been too low down to implicate the direct cerebellar tracts. Recalling Woroschiloff's experimental and his own clinical observations, Gowers concluded that the tract in question subserved cutaneous sensory impulses.

A year later than the appearance of Gowers' work, Hadden showed before the Pathological Society of London specimens of a symmetrical degeneration in the anterior part of lateral columns. There was, however, no degeneration of the posterior columns, nor, unfortunately, any history attaching to the case.

In 1884, Déjérine published a paper, based upon two tabetic cases, in which he denied any true systemic character to the lesion occupying the periphery of the antero-lateral column. It is, he said, merely a chronic superficial myelitis secondary to a chronic pial meningitis, and propagated by continuity of tissue from the overlying pia mater to the underlying nervous structures. He termed it part of an annular "méningo-myélite corticale à marche subaiguë ou chronique suivant les cas." Against this view Strümpell had spoken, and Westphal has since urged the objection, that in two cases in which the peripheral degeneration had been found and figured by him, there was no thickening or other anatomical sign of inflammation of the pia mater, certainly not a trace in the region covering the degenerated area.

It is difficult to see how a meningitis could give rise beneath it to the symmetrical comma-shaped degeneration recorded by Gowers, for the head of the inverted comma lies

removed from the periphery of the cord. And Hadden's case, if related to the others, exhibits the deeper portion of the tract suffering without participation by the peripheral. And yet stronger is the argument furnished by the observation of Bechterew, that a tract of fibres corresponding in position to a portion of the comma-shaped tract, develops apart from the rest of the lateral column.

Quite recently also further evidence in favour of the truly systemic nature of the lesion has been produced by Tooth. In a case of compression of the cord at the fifth dorsal vertebra, ascending degeneration had not only affected the posterior columns and the cerebellar tracts, but also areas situate at the periphery of the lateral columns near their junctions with the anterior. Still more lately has Westphal figured a border-degeneration (Rand-degeneration) in a case of diseased posterior columns reported at length in the 'Archiv für Psychiatrie'; and Strümpell in the same journal described and figured a very striking, although not uncomplicated example of it. The latter authority remarks, that there was no trace of concomitant meningitis; that the degeneration occupied the whole antero-lateral periphery from the cerebellar tract behind to the direct pyramidal at the lip of the anterior median fissure; and lastly, that we possess no hint as to the function of the fibres involved.

Not without reason, therefore, did Gowers publish, in the 'Neurologisches Centralblatt' of last March, a new note on his original case, adding that he had observed disease of the same tract in a couple of cases of tabes dorsalis.

To consider more fully the observation of Bechterew.

The original description by Türck, &c., of secondary degenerations of the pyramidal tracts, &c., acquired much greater weight when it became evident that the degeneration method and Flechsig's developmental method arrived at similar results. Flechsig, however, in the developing human cord, discovered no such tract as that found degenerated by Gowers. After distinguishing in the lateral column the crossed pyramidal and direct cerebellar tracts, he classed all the rest of the lateral column together as a "Vordere gemischte Zone." After distinguishing in the anterior column the direct

pyramidal tract, he classed all the rest of it together as the Vorderstrang Grund-bündel. His work yields no evidence of the tract marked out by the comma-shaped border degeneration in the lateral and anterior columns. Recently, however, from material investigated in Flechsig's laboratory by Prof. Bechterew, the latter has been able to find in the lateral columns a tract of fibres developing apart from the rest of the mixed zone, and corresponding to a portion of the tract demonstrated by the ascending degeneration of Gowers; the portion thus confirmed is that bounding the direct cerebellar tract.

But the developmental method does in reality confirm, completely and in its entirety, the tract described by Gowers. The middle of the 8th month of intra-uterine life is the epoch at which to look in the human offspring for such evidence. The developing spinal cord then shows the tract precisely as delimited by Dr. Gowers. At that period there are three tracts, of which the nerve-fibres have not yet acquired their medullary sheaths; they are the pyramidal, crossed and direct; the tract of Lissauer adjoining the posterior corner, the comma-shaped antero-lateral tract. Of these three tracts Lissauer's is the first to become medullated; the pyramidal, crossed and uncrossed, the last. The comma-shaped antero-lateral has an intermediate date. But although the fibres of the comma-shaped tract may be spoken of as roughly all becoming medullate, at the same epoch, that is not strictly true. The more anterior (ventral) lying fibres acquire the medullary sheath earlier than do the more posterior (dorsal); also those nearest the crossed pyramidal tract acquire it earlier than do those at the margin of the lateral column. The fibres latest to develop are those at the margin of the lateral column, just in front of the direct cerebellar tract.

The tract is not marked off from the cerebellar by a sharp line, but groups of its fibres lie islanded within the more anterior (ventral) portion of the latter.

Probably Bechterew observed the tract at a period when its more anterior and internal portions were already medullate. Such a supposition accounts for his description of it, and the discrepancy between his observations and those of Gowers.

In figures 1, 2, and 3, of the plate, I give the appearance of the tract in the upper dorsal and cervical regions of the spinal cord at the 8th month of intra-uterine life. Fig. 4 shows it in the cervical region at a later stage, where its appearance agrees better with the description by Bechterew.

Stated briefly, the anatomy of the ascending antero-lateral tract is as follows. In position it stretches throughout the lumbar, dorsal, and cervical regions, occupying in cross-section a comma-shaped area, the head lying in the lateral columns touching the ventral edge of the pyramidal tract, the tail extending along the edge of the antero-lateral column from cerebellar tract behind, to anterior median fissure in front. At the top of the cervical region where the cerebellar tract comes to lie more anteriorly (ventrally) and the crossed pyramidal reaches the margin of the lateral column behind the cerebellar, the comma tract besides occupying the same area as it does at lower levels, stretches as a thin band between the cerebellar and pyramidal tracts.

The tract does not degenerate below a point of injury, but does degenerate above it. After injury about the level of the last dorsal nerve-root, its degeneration does not reach higher than the upper part of the cervical enlargement; a large proportion of the fibres composing the tract must therefore be confined to the spinal cord itself. Bechterew believes the tract at its upper extremity enters the nucleus lateralis of the medulla oblongata; it must be remembered, however, that he only observed the more posterior lying portion of the tract.

As to experimental evidence of the antero-lateral tract, Langley, in the April number of this Journal, drew attention to the probability that many of the fibres in the more ventral portion of the periphery of the lateral column, though they degenerate upward, are not fibres belonging to the direct cerebellar tract. In the mouse, after injury to the spinal cord at the level of the 4th lumbar nerve-roots, an ascending degeneration in the posterior columns and in the peripheral portion of the lateral column occurs, which, at a distance of nine nerve-roots above the injury to the cord, presents the localisation represented in fig. 5 of plate. There certain fibres are degenerating in Goll's columns, and certain fibres in

the periphery of the lateral column, but not in that part of the periphery which, judging by analogy from the human and canine cord, belongs to the direct cerebellar tract. It is probable that the lesion was altogether too far down in the lumbar region to implicate the cerebellar tract, for I find nowhere good evidence of its degeneration. I imagine that the tracts degenerating at the level of the 7th dorsal nerve-roots (plate, fig. 5) are representative of the tracts that give in man the ascending degeneration in Goll's column, and the ascending antero-lateral degeneration of Gowers'.

At the level of the 5th cervical nerve-roots, no degenerating fibres that I could detect were present in the cord of the mouse. The animal had been killed on the twentieth day after the operation upon the lumbar cord. On the supposition, generally admitted, that fibres secondarily degenerating in the spinal cord undergo that process almost, if not absolutely, simultaneously throughout their length, it follows from the above that the tract in question does not everywhere pass unbrokenly from cord to brain, although presumably those of its fibres whose distal organ is in the upper dorsal and cervical regions, do do so. The portion of the tract passing upward from the lumbar region does not appear to reach further than the lower cervical region. With regard to the function of the tract, the original experiment by Woroschiloff, the degeneration in an ascending direction, the clinical observations of Gowers, and the results obtained by Bechterew, all point to a sensory function.

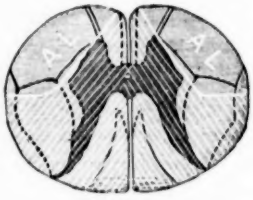
I have repeated Woroschiloff's experiment three times—on dogs instead of on rabbits—and have supplemented the usual indices taken as evidences of sensation by observations on the blood pressure, after the plan adopted by Miescher. The three experiments yielded the same results. I quote the second of them.

June, 17, 1886.—Dog, ♂, weight, 14½ kilos.

10 A.M.—Chloroform, later chloroform and ether mixed, administered.

Spinal cord exposed through posterior arch of 9th dorsal vertebra; the posterior half of the spinal cord divided with a galvano cautery; a Beer's cataract knife then pushed forward (downward) through the thickness of the cord twice, each time with its straight edge in the median line, the first time the oblique edge toward the right, the second time oblique edge toward left. (In this way the only portions of the cord at the point of operation that remained unsevered were the two

antero-lateral portions marked in the diagram by A L. A L.) Hæmorrhage stopped; wound covered by integuments; animal allowed to recover from anæsthetic.



Dorsal.

2 P.M.—Animal has recovered from anæsthetic, and has taken some food. Hind-limbs completely paralysed and flaccid; but pricking, pinching, and electric stimulation of the hind feet appear indubitably felt. The animal looks round to see what has touched him: hard pinching appears to cause discomfort, for the dog turns then quickly and angrily as if to bite.

2.15 P.M.—Chloroform again administered. Artificial respiration employed. Curare given by external jugular vein. The left carotid connected with a Ludwig's kymographion. The two sciatic nerves, right and left, prepared, and sheathed electrodes placed upon them. Time marker, seconds, and electric signal also registering upon drum of kymographion. Two pint Daniells in primary circuit of a Du Bois apparatus, for stimulation.

2.50 P.M.—Aortic blood-pressure as observed through left carotid is 11 mm. Hg.

2.55 P.M.—Stimulation for 3·5″ of right sciatic nerve, the secondary coil of the Du Bois (smaller) apparatus being at 10 cent. from the primary, causes blood-pressure to rise from 113 mm. Hg. to 127 mm. Hg. The curve of the rise as marked on the kymographion tracing shows two maxima, the earlier reaching 127 mm. Hg., the later—22″ later—reaching 124 mm. Hg.

2.58 P.M.—Stimulation, for 3″ of the left sciatic, with same strength of current, gives a similar rise of aortic blood pressure, showing again two maxima, an earlier of 129 mm. a later of 130 mm.

(These results were obtained several times over, and the experiment then concluded thus:)

3.40 P.M.—Abdominal aorta ligatured just below origin of lowest pair of lumbar arteries.

3.55 P.M.—Right sciatic stimulated with similar result to those recorded prior to ligature of aorta.

3.58 P.M.—Left sciatic gives similar result to right.

4.5 P.M.—Partial section of dorsal spinal cord turned into a complete division with the knife.

4.15 P.M.—Stimulation of sciatic nerves with secondary coil pushed over primary at full has no effect upon aortic blood pressure.

Thus sensory stimuli were able to initiate impulses that passed upward to the medulla through the portions A L of the cord at the ninth dorsal vertebra. Local vasomotor centres exist in the spinal cord, but that it was through these below the seat of operation that the rise of aortic blood-pressure

observed in the experiment was brought about, is negatived by the persistence of the reflex rise in the line of the blood-pressure tracing after cutting out from the general circulation both hinder extremities and the pelvic viscera. The immediate effacement of the blood-pressure rise on turning the partial into a complete section of the spinal cord, again confirms the same conclusion. I may add that in one experiment the splanchnics on each side were divided, and that the reflex rise in blood-pressure persisted as before, although the general blood-pressure had of course become lower.

A tract of small fibres at the junction of the lateral and posterior columns has recently been described by Lissauer as containing all the small fibres entering the cord from the posterior roots of the spinal nerves. In view of Gaskell's late researches into the functions of the small fibres of the nerve roots, this description by Lissauer acquires the highest interest. The more so to the pathologist, that the tract is frequently sclerosed in tabes dorsalis. The independent existence of Lissauer's tract has also been this spring affirmed by Professor Bechterew, pursuing the developmental method of research. He includes it in the lateral column as a special constituent of that column.

From my own observations I find distinct developmental confirmation of Lissauer's tract, both in man and in the dog. About the middle of the eighth month of intra-uterine life in man, and in the newly-born puppy, a tract of fine fibres wedged in between the lateral and posterior columns begins to become medullate. By the ninth month these fibres are fully medullate. Until they are medullated, very few medullated fibres are to be found in the substantia gelatinosa of the posterior horn; a fact which confirms Lissauer's view of the identity in part of the two sets of fibres. The amount of substantia gelatinosa and the size of the area occupied between posterior and lateral columns by the small-fibre tract appear to me roughly proportional also, where one is large, so also is the other, and *vice versâ*. Bechterew also observed this relation. But to consider the tract a part of the lateral column as Bechterew does, involves a somewhat strained nomenclature. If the tract observed by Bechterew—he gives no figures of it—

be really the same as that described by Lissauer and observable in my specimens, it seems to me distinctly within the posterior column.[1] Indeed, according to my own preparations, it would seem to extend along the surface of the posterior external column for a varying but sometimes (lower cervical region) considerable distance toward the posterior median fissure. Lissauer's tract of fine fibres is easily seen in the crocodile's cord, and Dr. Gaskell has pointed out to me that in that animal the periphery of the whole of the posterior column is occupied by a zone of similar fine fibres. I find, as did Bechterew, that Lissauer's tract runs through the entire cord from the middle of the sacral region upwards. For the dorsal and cervical regions it is shown in figs. 1, 2, 3 and 4.

Finally, to return for a moment to the discrepancies in the hitherto published descriptions of the tract, called by Gowers the ascending antero-lateral. They contrast strongly with the unanimity of all descriptions of the pyramidal tract. Both pathological and developmental evidence point to the ascending antero-lateral tract being, as is in reality of course the crossed pyramidal, a congeries of smaller tracts, not all attaining their full development absolutely simultaneously, and, though close together, yet fairly independently localised. On the other hand, that all subserve analogous purposes and connect distant spinal segments in analogous fashion, one can hardly fail to believe.

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[1] In the lower lumbar region, and at the level of the lower extremity of the pyramidal decussation, the tract of Lissauer certainly does encroach upon the limits of the lateral column. That the size of Lissauer's tract in cross-section does bear direct proportion to the amount of substantia gelatinosa is only roughly true. At the top of the cervical region, for instance, the tract of Lissauer is as large as in the lumbar swelling. Again, the tract as one follows it from the spinal cord into the medulla oblongata, in which latter it at first bears the same relation to the substantia Rolandi as it does to the substantia gelatinosa in the former, becomes less and less distinct upward, although the substantia Rolandi progressively enlarges.

Fig. 4

Fig. 2

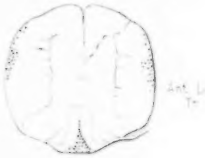

Fig. 5

Fig. 3

Fig. 1

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EXPLANATION OF PLATE.

Figs. 1, 2, and 3.—Cross sections of spinal cord at levels of 4th cervical nerve-root, of 2nd dorsal nerve-root, and of lower pyramidal decussation, respectively. From human fœtus at middle of eighth month. Coloured by Weigert's hæmatoxylin method, and magnified four diameters. Ad nat. del.

Fig. 4.—Similar to Fig. 1 above, but taken from the human fœtus of the ninth month.

The depth of shading represents the degree of development attained by the various tracts, the darker being the more completely grown. The only portions left white are the central grey substance of the cord and those tracts in the white matter whose fibres have not yet acquired medullary sheaths.

Fig. 5.—Spinal cord of mouse at level of 7th dorsal nerve, showing position of experimentally produced ascending degenerations.

Abbreviations used for the figures:—
Pyr. = pyramidal tract.
Cbll. = cerebellar tract.
Ant. Lat. Tr. = anterolateral tract.
L.'s Tr. = tract of Lissauer.

THE LATERAL RECESSES OF THE FOURTH VENTRICLE; THEIR RELATION TO CERTAIN CYSTS AND TUMOURS OF THE CEREBELLUM, AND TO OCCIPITAL MENINGOCELE.

BY J. BLAND SUTTON, F.R.C.S.,

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THE Pathological Society of London, on the evening of February 2nd, 1886, was concerned with the exhibition of a collection of intra-cranial tumours, which for richness and variety of material has probably never been equalled in the history of cerebral pathology. Among the specimens there shown, four especially arrested my attention. Two were exhibited as tumours in connection with the seventh nerves, the third and fourth as tumours of the cerebellum. The first two exhibited by Dr. Goodhart may be briefly described as follows: A tumour on each seventh nerve removed at a post-mortem examination in 1873. The case had been under the care of Dr. Wilks. The tumour on one side was as large as a pigeon's egg, whilst that on the other side was much smaller. The growths, which were of a fibro-sarcomatous nature, occurred in a woman, aged 26.

In the second case the tumour was of similar character, but unsymmetrical, and occupied the seventh nerve.

The third specimen was exhibited by Dr. Frederick Taylor as a fibro-sarcomatous tumour, growing out of one side of the flocculus of the cerebellum; it measured half-an-inch in length, was one-third of an inch in breadth, and a quarter of an inch in thickness, and gave rise to no demonstrable symptoms.

The fourth case, described by Dr. Beevor, was the brain of a man aged 40, with two tumours, one on either side of the medulla. The growth on the left side was of the size of a

bantam's egg. It compressed the cerebellum and pons Varolii, and had by pressure destroyed the fifth, seventh eighth and ninth nerves. The tumour on the left side was of the size of a hazel-nut, and occupied the angle between the flocculus, pons and medulla. It has pressed upon the seventh and eighth nerves (Fig. 4).

These four specimens presented a set of common characters which set me thinking. They were all situated in the immediate neighbourhood of the flocculus, and of the seventh cranial nerve; they presented the same naked-eye characters, and agreed histologically.

In Dr. Goodhart's first specimen the disease was symmetrical, and this symmetry is by no means infrequent in this class of cases. In Dr. Beevor's case the tumours were symmetrical.

In addition to the above features the following fact is somewhat remarkable. Among all the neoplasms exhibited on that noteworthy occasion there were no other specimens in any other part of the brain which resembled these tumours, except one also exhibited by Dr. Goodhart as a fibrous growth of the left fifth nerve; but this I shall later endeavour to show had its origin from the same source as the preceding tumours.[1]

At that time the question of symmetry and asymmetry of disease was occupying my leisure time, and the nature of these growths seemed a profitable subject for investigation, especially as the normal anatomy of the particular portion of the brain, for which these tumours have a predilection, has long been a favourite study with me.

Turning our attention to the normal disposition of parts in the neighbourhood of the flocculus we shall find, as represented in Fig. 1, that the cavity of the fourth ventricle is extended laterally by means of two tubular prolongations, known as the lateral recesses, which pass between the superior and inferior peduncles of the cerebellum to open in the subarachnoid space on either side. These recesses are in close relation with the flocculus, and with the facial, auditory, glosso-pharyngeal and vagus nerves. The fibres of the auditory nerve after leaving the floor of the fourth ventricle, lie on the anterior wall of the

[1] 'Many similar cases are recorded in periodical literature, and in the early volumes of the 'Transactions of the Pathological Society.'

recess. Prolongations of the choroid plexus pass through the recesses, and terminate in little villous masses at the base of the flocculus.

These lateral recesses, and the important relations they establish between the subarachnoid cavity and the ventricular system of the brain, has not received the attention they deserve,

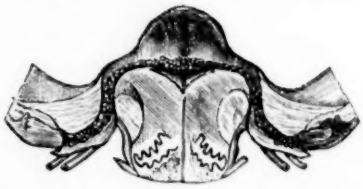

FIG. 1.—A transverse section of the fourth ventricle of the brain, showing the lateral recesses with their fringes of choroid plexus. The nerve filaments belong to the glosso-pharyngeal and vagus.

either from the anatomist or pathologist. The credit of discovering this connection belongs to Key and Retzius, who have given a full description of the part in their monograph, 'Studien in der Anatomie des Nervensystem,' Stockholm, 1875. In the Erste Hälfte, Seite 111 und 115, "Ueber der offenen Zusammenhang der Hirnventrikel mit den Subarachnoidalräumen," the account is prefaced by a historical review, in which due credit is given to preceding anatomists, Bockdaleck, Reichert, and Luschka, regarding the recesses, but their real nature was detected by Key and Retzius.

If the relations of the terminal extremities of the lateral recesses be inquired into, we shall find that they come into contact with that portion of the petrous bone indicated in the adult as the aqueductus vestibuli. A critical inquiry into this region discloses certain relations of no small degree of interest.

During the development of the internal ear, one of the most conspicuous features is a tubular prolongation, derived from, and remaining in communication with, the primary otic vesicle known as the recessus labyrinthi. This pierces the cranial wall, and becomes imbedded in the substance of the petrous portion of the temporal bone. Although very large in early fœtal life, it fails to grow at the same rate as the rest of the

labyrinth; at birth it is represented as a narrow passage, terminated by a bulbous end, lined by epithelium, and known as the saccus endolymphaticus, and is lodged in the bony canal named aqueductus vestibuli.

Although in the later periods of fœtal life the saccus endolymphaticus is shut from the general cavity of the arachnoid by the dura mater, nevertheless in early embryonic life this duct establishes a communication between the labyrinth and the

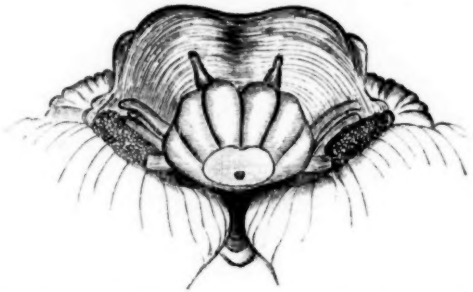

Fig. 2.—The pons and medulla represented somewhat diagrammatically, showing the tufts of choroid plexus emerging from the lateral recesses near the flocculus, between the seventh and eighth nerves anteriorly, and the glossopharyngeal posteriorly. (After Henle.)

general arachnoid cavity. This communication, according to Professor Owen,[1] was known to Cotunnius.[2] These connections are of interest, for in the lateral recesses we have a means whereby the auditory labyrinth of both sides are brought into temporary relation through the intervention of the fourth ventricle. The saccus endolymphaticus in animals is apparently a remnant of an intricate system of ossicles and ducts discovered by Weber[3] in the loach, carp and sheat-fish (*Silurus glanis*), where, through the intervention of a sinus impar lodged in the basi-occipital, the swim-bladder is brought into communication with the interior of the labyrinth.

Be this as it may, we have as matter of fact two tubular passages, leading from the cavity of the fourth ventricle to

[1] 'Anatomy and Physiology of Vertebrates,' vol. iii., p. 220, 1868.

[2] 'De Aquæductibus Auris humanæ internæ Anatomica Dissertatio:' 1774.

[3] 'De Aure et auditu Hominis et Animalium.' Lipsiæ, 1820, Part 1. 'De Aure Animalium Aquatilium.'

open into the general cavity of the arachnoid. These passages or ducts, like so many others in the body, are liable to become closed; in this case the obstruction is caused by a process of arachnoid occluding the outer end of the recess. The result of such occlusion is dilatation of the obstructed passage.

Besides stoppage of the recess this way, we must take into consideration the fringe of choroid plexus normally occupying its interior. It occasionally happens that the villi of the plexus enlarge, and thus constitute an obstruction, but more frequently they become the seat of a sand tumour, Psammoma. We thus get three distinct pathological conditions at the termination of the recesses. (1.) Cysts. (2.) Enlarged villi. (3.) Tumours.

(1.) It will be convenient to commence with the consideration of the cystic conditions. Virchow[1] figures a specimen bearing on the subject, and describes it as "a cystic hydrocele of the fourth ventricle with paralysis of the right facial nerve." Remnants of the choroid plexus were seen in the cyst. There was also hyperplasia of the pons Varolii and of the left hemisphere of the cerebellum. The case came from the Hospital of St. Hedwig.

The cyst was of the size of a cherry-stone and formed a projection on the surface of the brain, and clearly arose from the distension of that portion of the fourth ventricle known as the lateral recess. It pressed directly upon the facial nerve.

I have seen an exactly similar specimen in a brain which was dissected, whilst giving a demonstration on the anatomy of the brain to an ordinary dissecting-room class. On opening the fourth ventricle and endeavouring to expose the lateral recesses, the probe failed to make its appearance beside the flocculus, and on tracing the recess it was found to terminate in a *cul-de-sac* of the size of a nut. An example of the affection has been described by Recklinghausen[2] under the title of 'Doppelseitige Hydrocele der vierten Ventrikels.' The cysts were about the size of cherries. The author refers to Virchow's case quoted above. Between such simple cysts

[1] 'Pathologie des Tumeurs,' vol. i. p. 180.

[2] Virchow's 'Arch.,' Bd. xxx. s. 374.

as these and the tumours represented in Fig. 4 is a variety of cyst with very thick walls. The following will serve as a typical example.

The specimen is preserved in the museum of the Middlesex Hospital, and is of great interest, inasmuch as it was obtained by Sir Charles Bell, and the history of the patient, with a drawing of the parts, is given in his work on the 'Nervous System:'—

"The left Lobe of a Cerebellum and the Pons. Attached to the inferior surface of the left peduncle of the cerebellum close to its junction with the pons is a tumour the size of a pigeon's egg, consisting of a cyst with a distinct lining membrane, invested outside by a layer resembling convoluted brain substance. When recent, it was filled with a fluid the

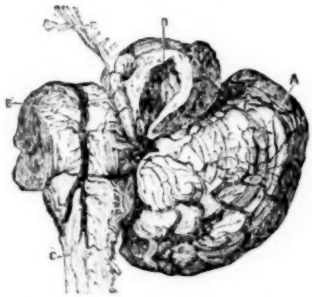

FIG. 3.—The under-surface of the cerebellum, showing a cystic tumour of the size of a pigeon's egg, involving the fifth, seventh and eighth nerves. (After Sir Charles Bell.)

colour of urine. The fifth nerve, attenuated and flattened, appears to issue from the fundus of the tumour, and can be traced along its walls up to within half an inch of its origin. Both the portio dura and mollis of the seventh nerve are lost in the tumour from within a quarter of an inch of their origin as far as the meatus internus."

(2.) From the cysts we must now pass on to the consideration of the tumours occupying this particular region of the brain. In doing so, one typical case will be described, and we shall then deal with the question of ætiology. As the type, I select that shown in the accompanying drawing, Fig. 4. It

is the same case already referred to as being exhibited at the Pathological Society, by Dr. Beevor. Through his kindness and the courtesy of Dr. Ferrier I have been allowed to re-examine and figure the specimen.

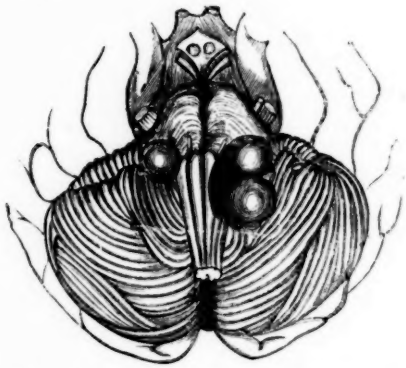

FIG. 4.—The under surface of the cerebellum. On the right side a large tumour is seen compressing the pons medulla, 5th, 7th, 8th, 9th, and 10th, nerves. On the left side a similar but much smaller growth is seen.
(Diagrammatic.)

Examined microscopically, this neoplasm conformed in all respects to those singular growths so frequently found of small size, and so often symmetrical, in the choroid plexuses of the lateral ventricles, and known as psammomata. Their structure is often described as fibro-sarcomatous, because they consist of fibrous tissue sprinkled everywhere with round cells. But their most distinguishing feature is the deposit in them, often in concentric masses of gritty particles, sometimes in considerable quantity, imitating in this way the structure of the pineal body. Chemically, the earthy matter is made up of carbonate and phosphate of lime.

When we remember the extreme frequency of these tumours of small size, in connection with the choroid plexuses of the ventricles, not only in man but also of the lower animals, especially horses, their occasional occurrence in larger masses, and then recognised as psammomata, both in the brain and connected with the membranes of the spinal cord, and whenever they occur they are related to the

arachnoid or its choroid plexuses, there need be little hesitation in ascribing the origin of the curious tumours described in the commencement of this paper to the cluster of villi situated in the angle formed by the flocculus, pons, and medulla. They agree with psammomata of the choroid plexus in the following points:—structural mimicry, symmetry of position, relation to arachnoid, and the age at which they occur, and the few clinical symptoms usually manifested by them.

(3.) It has long been a recognised fact, as Meckel pointed out in 1812, that cranial meningocele is by far the most frequent in the occipital region: in seventy-five cases tabulated by Laurence, fifty-three occupied this region. Another important circumstance connected with this deformity in the occipital region is the absence, or rudimentary condition, of the cerebellum.

All the cases of occipital meningocele or encephalocele which I have had opportunity of examining, serve to convince me that the cyst is in reality a distension of the cavity of the fourth ventricle. The accompanying drawing (Fig. 5) serves to illustrate this view of the matter.

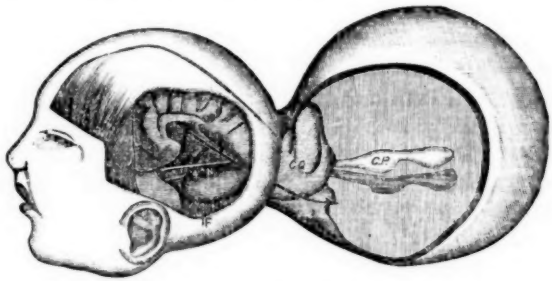

FIG. 5.—A ventriculo-meningocele. The tissue lining the cyst is continuous with the ependyma of the fourth ventricle. F. The flocculus (usually mistaken for a rudimentary cerebellum.) 4th N. The fourth nerve. C.Q. The corpora quadrigemina. C.P. Choroid plexus projecting into the cyst. The arrow lies in the Sylvian aqueduct. (From 'Path. Soc. Trans.' vol. xxxiv.)

The details of the case may be briefly summarised as follows:—

The child survived its birth six weeks;[1] when it died, the

[1] 'Path. Soc. Trans.' vol. xxxiv. plate iii.

cyst was as large as its head. In this specimen the lining membrane of the cavity is continuous with the walls of the fourth ventricle, and the cerebral matter projecting into it is probably the corpora quadrigemina; this view is strengthened by the fact that the fourth nerve is connected with it. The arrow indicates the *iter a tertio ad quartum ventriculum*. The fringes floating in the interior of the cyst represent the choroid plexus of the fourth ventricle. When I described this specimen originally, the cerebellum was said to be rudimentary, and this is the usual account given by those who have recorded cases of this kind. This is not absolutely correct, the cerebellum is absent, and the portion of brain attached to the medulla is, as a matter of fact, the flocculus, or sub-peduncular lobe.

My attention was first directed to this matter by reading a careful and suggestive paper by Cleland[1] in which the following note occurs. "The flocculus, though always described with the cerebellum, is very well understood by those who have studied its development to be totally unconnected with that organ, being developed from a lateral outgrowth of the floor of the third cerebral vesicle, while the cerebellum is developed further forward from the foremost part of the roof of that vesicle."

This renders the question one capable of interpretation, for we have already seen that occlusion of one recess leads to cystic dilatation of the affected side. If both recesses become obstructed, it must necessarily lead to dilatation of the whole ventricular cavity, and occurring in early embryonic life, hinders the proper development of the cerebellum. Such a dilatation bears the same relation to a meningocele proper, as a syringo-myelocele bears to a spinal meningocele.

That this view offers a reasonable explanation of ventriculo-meningoceles in this situation, I venture to think few would doubt.

Virchow has described in his work on Tumours, in the part devoted to cysts, that occasionally one of the posterior cornua of the lateral ventricles becomes obstructed at the point where

[1] "Spina Bifida, Encephalocele and Anencephalus." 'Journal of Anat. and Phys.' vol. xvii. p. 257.

it joins the general ventricular cavity. As a consequence, the part behind dilates into a large cystic cavity.

If this be true of the cornua of the lateral ventricles,[1] and cystic dilatation of the fourth ventricle may arise from obstruction to the lateral recesses, we can extend the notion

FIG. 6.—The fourth ventricle from a case of occipital ventriculo-meningocele. The cerebellum is absent. The foliated mass represents the flocculus or sub-peduncular lobe.

further, and maintain that ventriculo-meningoceles, connected with the lateral and third cavities of the brain, may arise from obstruction to the aqueduct of Sylvius, occurring in early embryonic life, in the same way that it has been made to account for some forms of hydrocephalus.

SUMMARY.

The contents of this paper may be stated in an abstract form as follows:—

1. Closure or absence of the lateral recesses of the fourth ventricle, in early embryonic life, leads to dilatation of the third cerebral vesicle, and give rises to a ventriculo-meningocele, projecting at the occiput.
2. If the lateral recesses become occluded after birth, a local cyst or cysts result.
3. The fringes of choroid plexus occupying the recesses may enlarge and form a villous tumour.
4. Such a tumour forms an excellent nidus for lime salts, and then become recognised as Psammomata.

[1] Dilatation of the posterior cornua of the lateral ventricles of the brain are not uncommon.

ON CERTAIN QUESTIONS RELATING TO THE URINOLOGY OF THE INSANE.

BY ERNEST BIRT,

Senior Assistant Medical Officer, West Riding Asylum, Wakefield.

In a section of an important work,[1] published in 1884, Dr. W. Zuelzer treats of changes in the composition of the urine, associated with various conditions of altered metabolism in the nervous tissue.

He maintains that, from the nervous tissue, when in a state of lowered irritability, the delivery of material is augmented, and that it is lessened in conditions of exalted irritability. Further, that each of those series of conditions is, in respect to the tissue-change, differentiated by urinary qualities peculiar to it, and of such kind that, in depression conditions, (traumatic or pathological destructive brain-lesions; chloroform, ether, morphia, narcosis, &c.), the phosphoric and glycerinphosphoric acids[2] of the urine are increased; whereas, excitation conditions (as induced by strychnia, phosphorus, alcohol in small doses) are attended by a diminished amount of those products in the urine.

To illustrate the points claimed by Zuelzer, the following three analyses are selected from those contained in his work:—

Chloroform narcosis of 30 minutes' duration, for excision of adenoma. The urine voided immediately before the operation contained—[3]

N	P_2O_5	R
0·2648	0·076	28·7
	0·003	1.1

[1] 'Untersuch. über die Semiologie des Harns,' S. 57, u.ff.

[2] Also the potash and lime salts; but these are beyond the scope of this paper so far as estimation of the basylous element is concerned.

[3] Throughout this paper the quantities found by analysis are expressed in grammes, or parts of a gramme. Where two series of figures are given under

An hour after the operation—

N	P_2O_5	R
0·38	0·173	45·5
	0·049	12·8

Morphia administered internally. The night urine of 21st–22nd July, contained—

4·96	0·89	17·9

0·02 g. morphia taken during evening of 22nd. Night urine of 22nd–23rd July contained—

4·85	1·218	25·1

Strychnia Experiment on Dog.

October 29th–30th.—Urine of—

2·2	0·358	16·2

October 30th.—0.002 g. strychnia subcutaneously.

During the spasms (a portion lost)—

0·15	0·011	7·3

October 30th–31st.—Evening and early morning,

1·74	0·164	9·4

October 31st.—Forenoon,

1·96	0·279	14·2

Mid-day,

3·43	0·58	16·9

Zuelzer further remarks that the alteration in the composition of the urine, associated with the first mentioned series of conditions, brings the relationship between the existing amounts of phosphoric pentoxide and nitrogen into more or less close accord with that which obtains in the nervous tissue itself, and with that present in the urine of animals (dogs) fed on brain substance.

According to his estimates, the normal gravimetric proportion of the P_2O_5 to the N, in the 24 hours' urine of an adult, is 18 or 20 to 100. In blood, the mean proportion is as 4 : 100; in muscle, 15 : 100; in brain and other nervous

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the heading P_2O_5, the first indicates the phosphoric acid in combination with alkalies and alkaline earths, the second, the glycerinphosphoric acid. The numbers under R denote the gravimetric ratio of the respective P_2O_5 to 100 N.

organs, which contain the greatest amount of lecithin, 45 : 100.

With regard to the glycerinphosphoric acid, concordant and definite estimates of the maximum quantity, present in the urine under normal circumstances, are wanting. In the 24 hours' urine of a healthy adult, Zuelzer found 0·06 gramme = 2·3 p.c. of the total P_2O_5, but he gives no numerical generalization upon the point.

Hoppe-Seyler[1] describes its occurrence in "very small quantity in normal urine."

Lépine and Eymonnet[2] place the normal amount at 0·25 to 100 N, or about 1 p.c. of the total P_2O_5.

These latter observers also have noted an increase in the renal excretion of the phosphorus compounds—particularly the glycerinphosphoric acid—as a result of gross cerebral lesions, epilepsy, and use of chloral or bromides. Thus in a case[3] of hæmorrhage into the external capsule and outer part of the lenticular nucleus, the urine excreted during the first six hours contained, per litre,

N	P_2O_5	R
2·5	0·54	21·6
0·0268	1·07	

"Forty-eight hours later, the proportion was normal."

With the view of ascertaining whether the above described urinary characteristics are commonly present in certain of the neuropathic, and psychopathic states met with in asylums, I undertook the subjoined analyses.[4] Especially, it seemed to

[1] 'Handbuch der Phys. u. Path. Chem. Anal.' 1883, S. 116.

[2] 'Comptes Rendus des Séances de l'Acad. des Sciences,' t. xcviii., 1884, No. 4, p. 239.

[3] Loc. cit.

[4] It may be well to designate the methods employed. In all cases the nitrogen has been determined in a Knop-Wagner's azotometer, by the process described in Zuelzer's 'Manual on Urinary Analysis,' using Dietrich's correction tables for absorption and weight. Zuelzer calculates that with this apparatus the hypobromite disengages the N to within 0·1, or at most 0·2 p.c. of the existing quantity, unless the urine contain much uric acid—2 p.c., or less, of the N may then remain in combination. With Hüfner's apparatus the deficiency is stated to be 7 p.c. The most favourable result obtained by use of the improved Simpson and O'Keefe's method left 0·09 p.c. combined (Cranstoun Charles).

The phosphoric acid united with alkaline earths has been estimated by precipitation with ammonia, and titration with standard uranic solution.

That in combination with alkalies has been similarly titrated, after precipita-

me that such investigations might throw some light upon the nervous metabolic processes underlying those transient hemiplegiæ, or monoplegiæ, with or without stupor, which frequently may occur during the course of general paralysis, without leaving any macroscopic clue to their origin.

CASE I.—General paralysis, 2 years' duration. F. S., male, 43, habitually elated, boastful, the imaginary possessor of millions of pounds, three wives, &c. Was able to walk about until October 22, 1884; stupor, with rigidity of all the limbs, developed early on that morning.

The urine drawn at 1 P.M. (excretion of about 15 hours), contained—

N	P_2O_5	R	REMARKS.
10·69	2·481	23·2	
	0·1805	1·6	Max. axillary temp. 39·1°.
From 1 P.M. to 10 P.M.			
4·231	1·2659	29·9	
	0·0188	0·4	
From 10 P.M. to 9.30 A.M., *October 23rd.*			
7·461	2·5344	33·9	38·4°.
	0		
9.30 to 6 P.M.			
5·337	1·6898	31·6	
	0		
6 P.M. to 11 A.M., *October 24th.*			
9·24	3·024	32·7	Emerging from stupor: recognises attendant and calls him by name. Head and eyes persistently turned to right. Left limbs flexed and rigid: right limbs fairly under control.

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tion by ammonio-magnesian solution—the known quantity of earthy phosphates being subtracted from the result.

For ascertaining the amount of glycerinphosphoric acid I have nearly always employed the indirect method—prolonged boiling of the urine with nitric acid, rendering alkaline with ammonia, precipitating by the magnesian solution, titration, and subtraction of the known amount of alkaline and earthy phosphates. The direct methods (Lépine, Hoppe-Seyler, Sotnischewsky) comparatively are complicated, and involve a great expenditure of time.

Repeatedly, when this organically combined acid has existed in traces too small to be measured with certainty by titration, the microscope has enabled me to detect it in the form of a few crystals of triple phosphate.

11 A.M. to 6 P.M.

N	P_2O_5	R	Remarks.	
7·357	1.2	16·3		39·4°.
	0·12	1·6		

3 A.M. to 9.30 A.M., *October 25th.*

6·15	0·929	15·1		
	0·103	1·6		38·7.

From 6 P.M. to 10 A.M, *October 26th.*

11·82	1·715	14·5		37·6°.
	0			

6 P.M. to 9.30 A.M., *October 27th*

10·097	1·1787	11·6	Left hemiplegia persists: less rigidity in palsied limbs. 36·5°.	
	0			

11 A.M. to 6 P.M., *October 29th.*

	0·441			37·8°.
	0·0784			

October 30th.

10·703	0·9047	8·4		38·4°.
	0			

In this case, the large absolute quantity of the glycerinphosphoric acid on the first day, and the increasing elimination of the inorganically combined P_2O_5 during the period of stupor, are very prominent facts.

They certainly are independent of the food, for during those days the patient took only small quantities of milk, beef-tea, and egg—articles which he ingested freely when the stupor had passed off. Doubtless, the muscular tension and the pyrexia may have been the cause, to some extent, of the augmented excretion of the latter mentioned P_2O_5; but these conditions had not disappeared when the P_2O_5 elimination had permanently fallen much below the healthy standard.

The left limbs became flaccid, and remained paralysed till death on November 7, 1884.

There was no trace of focal lesion in the encephalon—beyond old adhesions of pia to cortex.

In the next two cases there exists a notable discharge of glycerinphosphoric acid in connection with the occurrence of paralytic motor phenomena, which contrasts strongly with

the absence of that compound when the patients had regained, what may be considered, their normal state.

II.—General paralysis, 4 years' duration. A. F., female, 40, quiet, demented, cheerful and clean.

October 10th, 1885.—After brief prodromal emesis, right brachial monoplegia, with speechlessness, occurred to-day. No extra impairment of consciousness; understands what is said, and responds, otherwise than verbally. Paralysed limb rigidly semi-flexed at elbow.

The urine drawn 12 hours after onset of attack, contained [1]

N	P_2O_5	R	Remarks.
3·298	0·105	3·2	
	0.644	19·5	
	0·028	0·8	

October 14th.—12 hours ending at noon.

8·514	0·1345	1·5	Remains speechless. Limb less rigid: slight return of voluntary power.
	0·7768	9·1	
	0·013	0·1	

October 22nd.—24 hours ending at 8 A.M.

10·289	0·272	2·6	Four convulsive attacks yesterday: none since 2 gr. chloral were given. Convulsions were general: head and eyes to right. Patient can nearly articulate "Yes." Limb continues paralysed.
	1·559	15·1	
	0		

October 24th.—12 hours ending at 10 P.M.

4·43	0·0168	1·05	No more fits.
	0·1368	9·8	
	0·0052	0·1	

February 26, 1886.—1 A.M. to noon.

2·514	0·112	4·4	No fit since October. Completely recovered use in limb. Speech partly restored.
	0·272	10·8	
	0		

February 27th.—1 A.M. to noon.

2·77	0·108	3·8
	0·288	10·3
	0	

[1] The first (topmost) group of figures shows the amount of P_2O_5 in combination with alkaline earths; the second, that united with alkalies; the third represents the glycerinphosphoric acid. This order is followed in all cases where the three series occur in this paper.

III.—General paralysis, 5 years' duration. E. C.,[1] female, 40. Very demented and dirty, fairly quiet, thoroughly self-satisfied.

November 27th, 1885.—Left brachial monoplegia came on in night. No stupor, convulsion, nor speech loss.

Urine passed at noon and 6 P.M. contained

N	P_2O_5	R	Remarks.
1·055	0·0728	6·8	
	0·084	7·9	
	0·0056	0·5	

November 28th.—Night and forenoon excretion.

2·277	0·0748	3·2	Recovered free use in limb yesterday evening.
	0·2912	12·7	
	0·2329	10·2	

December 7th.—Night and forenoon (part lost).

2·967	0·246	8·2	No further attack.
	0·126	4·2	
	0		

February 8th, 1886.—Night and forenoon.

2·8256	0·27	9·55	Has remained in usual state.
	0·09	3·22	
	0		

February 9th.—Night and forenoon.

2·838	0·2275	8·01
	0·0875	3·08
	0	

February 10th.—Night and forenoon.

3·001	0·26	8·66
	0·025	0·83
	0	

So far as my researches extend, one does not constantly find an excretion of glycerinphosphoric acid following the convulsive attacks of general paralysis. Similarly in epilepsies, even when an enormous series of fits rapidly proceeds to a fatal issue.

IV. General paralysis, unknown duration. J. W., male, 38.

February 19th, 1886.—Severe general convulsions from noon until 1.10 P.M. Deep coma throughout. Ten minutes before convulsions ceased, 2·7 grammes of chloral per rectum.

Urine drawn at 10 P.M. = total excretion since convulsions began, contained

N	P_2O_5	R	Remarks.
2·393	0·14	5·8	
	0·042	1·7	
	0		

February 20th.—12 hours ending 10 A.M.

4·003	0·0725	1·8	No further convulsion. Recovered consciousness this morning.
	0·7395	18·4	
	0·0195	0·4	

V.—General paralysis, 2 years' duration. A. H., female, 35.

February 10th, 1885.—Two strong convulsive fits, from which quickly recovered.

Urine drawn 3 hours after the fits contained

0·919	0·045	4·8
	0·095	10·3
	0	

April 23rd, 1885.—Convulsed in forenoon. Urine drawn at 1.30 P.M.

0·891	0·066	7·4
	0·069	7·7
	0	

Next 2 hours

0·531	0·0		No fit since morning.
	0·1004	18·9	
	0		

VI.—Chronic mania, 21 years. Epileptiform seizures, 2 months. No permanent contracture or paralysis. M. A. P., female, 60.

January 29th.—Convulsed for 2½ hours—the spasms being strong and mainly limited to left side. 1·5 gramme chloral per rectum.

Urine drawn near end of attack contained

1·252	0·0786	6·2
	0·2824	22·5
	0	

[1] This patient died in April, 1886. There was no local softening, nor could the naked eye detect any appearance to account for the monoplegia.

January 30th.—Urine of next 7 hours contained

N	P_2O_5	R	Remarks.
1·858	0·0982	5·2	No further convulsion.
	0·06	3·2	
	0·0109	0·5	

June 16th, 1885.—Fits recurred, with scarcely any intermission, for a period of 4 hours, ending at 3 A.M. 8 grammes of chloral, per rectum, during that time.

Urine passed at 2 P.M. (two previous evacuations lost) contained

	0·01	
	0·0225	
	0	

October 13th, 1885.—Strong convulsions from 5 P.M. to 7.15 P.M. 1·5 grains chloral at 7 P.M.

Urine from 6 P.M. till noon, *October 14th*, contained

2·533	0·0723	2·8
	0·1034	4·08
	0·0103	0·4

VII.—Epilepsy, many years. A. M., female, 35. Much demented, but clean and useful, corpulent.

January 4th, 1886.—9 severe fits in 12 hours ending 5.30 A.M. No great prostration or prolonged stupor.

Urine of night and forenoon contained

3·18	0·288	9·05	Has daily taken 4 gr. alkaline bromide for more than a year.
	0·216	6·7	
	0		

January 5th.—Three fits from 1 A.M. to noon.

Urine of night and forenoon contained

3·22	0·0784	2·4
	0·294	9·1
	0	

VIII.—Epilepsy, since puberty. S. T. P., female, 22. Demented, irascible and pugnacious. Movements, spastic and sprawling. Great prostration after fits, feeble health, spare.

December 21st, 1885.—Eight fits yesterday; 2 in night. 3 grammes of chloral in night.

Urine of night and forenoon.

2·87	0·27	9·4	For many months has taken 3·5 grammes of potassium bromide each day.
	0·4212	14·6	
	0·0243	0·8	

December 22nd.—Such as could be collected during 24 hours, ending at 2 P.M.

N	P_2O_5	R	Remarks.
3·53	0·3012	8·5	No more fits or chloral.
	0·3102	8·7	
	0·0094	0·2	

December 25th.—Three fits from 6.30 A.M. to 9.10 A.M. Urine drawn at 3.30 P.M. contained

1·709	0·2244	13·1	
	0·4048	23·6	
	0·0176	1·02	

January 3rd, 1886.—Urine from 4 A.M. to 10.30 A.M.

1·83	0·0925	5·05	No fit for 30 hours.
	0·3071	16·7	
	0·0074	0·4	

IX.—Epilepsy, since puberty. S. H. B., female, 36. Very demented, sluggish and awkward. Corpulent.

For five days the urine was collected in the night and forenoon.

January 23rd, 1886.

2·545	0·13	5·1	Two fits in forenoon. No medicine.
	0·403	15·8	
	0		

January 25th.

1·593	0·0825	5·1	Three fits in night.
	0·2425	15·1	
	0		

January 26th.

2·21	0·14	6·3	One fit in morning, one in afternoon.
	0·35	15·8	
	0		

January 27th.

2·027	0·185	9·1	No fit.
	0·2925	14·4	
	0		

January 28th.

3·211	0·165	5·1	No fit.
	0·78	24·2	
	0		

X.—Epilepsy, since puberty. M. P., female, 33. Quiet, industrious, imbecile. Fair general health.

January 16th.—Five severe fits in the afternoon; 6 during the preceding night; 3 in the following night.

Urine from 7.30 P.M. to noon, *January 17th*, contained

N	P_2O_5	R	Remarks.
3·779	0.38	10·0	Takes 3 gr. of bromide daily.
	0·4192	11·0	
	0·0633	1·6	

January 29th.—Urine of similar period.

10·812	0·315	2·9	Two fits in night. None for 3 preceding nights.
	0·735	6·7	
	0		

January 30th.—Urine of similar period.

3·099	0·2142	6·9	No fit.
	0·4488	14·4	
	0·0102	0·3	

XI.—Epilepsy, many years. M. M., female, 30. Much demented, sluggish, feeble and obese. Average number of fits, 2 weekly.

Seventy-seven severe fits during the 48 hours ending at noon, January 31st, 1886. 9 grammes of chloral were administered in that time. Death occurred shortly before midnight, at the onset of a fit—convulsions having been absent for several hours previously;[1] but a fatal termination from pulmonary engorgement and œdema having appeared inevitable for many hours.

The urine drawn in the evening of January 30th, and on the morning of January 13st contained

2·142	0·085	5·96
	0·391	18·25
	0	

[1] It is a noteworthy fact that, in this Asylum, during the past seven years the only cases in which chloral has failed to arrest convulsions before irremediable pulmonary hypostasis has developed, have been those in which the patient has not been taking a bromide, continuously, for a long period. The observation extends to 13 males and 17 females, who died in the epileptic status. Two of the male patients commenced the bromide just after coming to the asylum. Of them, one died within a week, the other within a fortnight. Two of the females terminated a long course of bromide a month, and five weeks, respectively, prior to the commencement of the fatal series. None of the other patients had undergone bromide treatment for many months, or years, before the last outbreak of fits occurred.

XII.—Epilepsy, 18 years. B. D., female, 21. Imbecile, robust general health.

January 29th, 1885.—59 strong fits last night—only 2 after a second 2 gramme dose of chloral per rectum.

The urine passed at bedtime of the 28th January, 1885, before the fits came on, contained

N	P_2O_5	R
2·208	0·2508	11·3
	0·3225	14·6
	0·0358	1 6

January 29th, 1885. Urine drawn at 11 A.M.

1·765	0·0806	4·5
	0·018	1·01
	0	

Death took place a few hours later.

Professor Lépine and M. Jacquin have recorded[1] some interesting observations concerning the P_2O_5 excretion in certain cases of essential epilepsy and of epilepsy from alcoholism. They found the proportion of the P_2O_5 to the N much below normal on the days between the fits—once as low as 8·6 p.c. In those patients the proportion notably increased immediately after a fit, the rise being absolute, and chiefly due to an increase of the earthy phosphates. An augmentation of the earthy phosphates was also noted when the patients had merely experienced sensations of a fit being imminent, or had undergone an attack of vertigo.

Those special features cannot be definitely traced in the foregoing analyses—Cases IV. to XII. Frequently, indeed, the ratio of earthy to alkaline phosphates is there excessive; but that anomaly is almost constantly owing to the remarkably small absolute quantity of the latter. Thus, reference to IV., VI., VII., and XII. shows (not without exceptions) an extraordinary failure of the alkaline phosphates in the urine first collected after the nervous discharge. A like phenomenon is present in some of Lépine's analyses. It is necessary to observe, however, that Lépine investigated the whole 24 hours' urine, for several days in succession; consequently a full comparison of my analyses, cited above, with those of Lépine, cannot be made.

[1] 'Revue Mensuelle de Méd. et de Chir.' tome iii. 1879, Nos. 9 et 12.

In the next two cases the 24 hours' urine was collected, save on certain days indicated. Here, also, are some manifest disproportions in the amount of the phosphates; universal in the first case, greatest during the acme of the convulsive period in the second, but also existing on days[1] when no objective evidence of a fit was present. (It is not known whether auræ occurred at those times.) This disparity is, again, mainly the result of diminished alkaline phosphates.

It should be noted that the second patient took less food after the fits commenced, and then suffered from malaise, lassitude, and headache, which prevented her from working.

XIII.—Epilepsy, many years. M. M., female, 41. Much demented, excitable, quarrelsome, and noisy. Feeble health; exceedingly macilent.

January 30–31*st.*

N	P_2O_5	R	Remarks.
3·894	0·315	8·08	One fit on Jan. 30.
	0·504	12·9	
	0·0157	0·4	

January 31*st*–*February* 1*st.*

3·505	0·368	10·4	No fit.
	0·46	13·1	
	0·092	2·6	

February 3–4*th.*

5·403	0·456	8·4	One fit on Feb. 3.
	0·57	10·5	
	0		

February 4–5*th* (some lost).

4·584	0·264	5·7	No fit.
	0·286	6·2	
	0		

February 5–6*th.*

4·0492	0·371	9·1	One fit on Feb. 5.
	0·2915	7·1	
	0		

February 6–7*th.*

4·415	0·3444	7·8	No fit.
	0·3444	7·8	

XIV.—Epilepsy, since puberty. C. H., female, 35. Not

[1] See Feb. 12, 13, 16, 18–20, and 27. On March 1 and 4 the ratio of earthy to alkaline phosphates is quite normal.

much demented, never displays great excitement, is a useful ward cleaner. Fair general condition. Fits usually occur at the catamenial periods.

February 10–11*th.*

N	P_2O_5	R	Remarks.
6·544	0·387	5·9	No fit for three weeks.
	1·29	19·7	
	0·043	0·6	

February 11–12*th.*—(Part lost).

4·925	0·36	7·3
	0·88	17·8
	0	

February 12–13*th.*

5·163	0·343	6·6
	0·686	13·2
	0	

February 13–14*th.*

6·7155	0·462	6·7
	1·188	17·6
	0	

February 14–15*th.*

8·451	0·532	6·2
	1·292	15·2
	0	

February 15–16*th.*

5·544	0·432	7·7
	0·864	15 5
	0	

February 16–17*th.*

5·7	0·324	5·6
	0·9	15·7
	0	

February 17–18*th.*

7·734	0·511	6·6
	1·095	14·1
	0·073	0·9

February 18–19*th.*

7·1	0·474	6·6
	0·948	13·3
	0	

February 19–20*th*.—(A portion lost.)

N	P_2O_5	R	Remarks.
5·465	0·441	8·06	
	0·686	12·5	
	0		

February 20–21*st*.

7·23	0·346	4·7	
	1·314	18·1	
	0		

February 21–22*nd*.

9·089	0·489	5·3	
	1·876	20·6	

February 22–23*rd*.

5·836	0·504	8·6	
	1·152	19·7	
	0		

February 23–24*th*.

5·608	0·395	7·04	
	1·187	21·1	
	0		

February 24–25*th*.

8·88	0·408	4·5	
	1·292	14·5	
	0		

February 25–26*th*.

9·018	0·528	5·8	
	1·254	13·9	
	0		

February 26–27*th*.

8·06	0·504	6·2	No fit hitherto.
	1·008	12·5	
	0		

February 27–28*th*.

11·127	0·648	5·8	One fit.
	1·8	16·1	
	0		

February 28*th*–*March* 1*st*.

8·42	0.497	5·9	One fit.
	1·704	20·2	
	0		

March 1–2*nd*.—(Part lost.)

4·897	0·424	8·6	Five fits.
	0·656	12.9	
	0·106	2·1	

March 2–3rd.—(Much lost.)

N	P_2O_5	R	Remarks.
2·649	0·216	8·1	Seven fits.
	1.36	13.5	Glycosuria.
	0		

March 3–4th.—(Much lost.)

4·41	0·253	5·7	Three fits.
	0·897	20·3	Trace of sugar.
	0		

March 4–5th.—(Much lost.)

5·239	0·42	8·01	One fit.
	0·595	11·3	No sugar.
	0		

In a case of recurrent mania, some details of which are annexed, the alternating mental phases were to some extent characterised by change in the P_2O_5 renal excretion—the ratio of that to the N was constantly lower when excitement was absent. No organically combined P_2O_5 was found while the mental affection ran its usual course. As soon, however, as a depression condition became established (partial collapse from peritonitis, sopor induced by morphia), a large elimination of glycerinphosphoric acid occurred. Muscular activity and alimentation can each be excluded from the production of this phenomenon, because the former was reduced to the oppressed exercise of the circulatory and respiratory muscles, with infrequent emesis, and the latter was totally prevented by the inverted action of the stomach.

Analysis was made from all the urine that could be obtained in the 24 hours; on some days, little, or none, was lost. The earthy phosphates were not separated from the alkaline.

XV.—Recurrent mania, 3½ years. N. M., male, 44. When maniacal, incessantly restless, noisy, gay, and mischievous; almost sleepless for several days. During calm periods, industrious and orderly. Feeble health; heart fatty, spare condition.

September 30th, 1884.

N	P_2O_5	R	Remarks.
7·057	1·937	27·4	Maniacal for three days.
	0		

October 2nd.

N	P_2O_5	R	Remarks.
8·5	1·7	20·0	
	0		

October 4th.

6·518	1·4124	21·6	Mania subsiding.
	0		

October 8th.—10 A.M. to 5 P.M.

1·951	0·472	24·1	Nearly free from excitement.
	0		

October 9th.—Night urine.

1·756	0·315	17·9	
	0		

October 13th.

7·446	1·585	21·2	Quite tranquil.
	0		

October 14th.

3·481	0·5372	15·4	
	0		

October 15th.

7·669	1·116	14·5	
	0		

October 16th.

5·385	0·81	15·04	
	0		

October 17th.

6·9	1·302	18·8	
	0		

October 21st.—6 A.M. to 11 A.M.

3·966	0·8613	21·7	Relapse commenced yesterday. Peritonitis developed in night 0·016 gr. morphia, subcutaneously, at 9.30 A.M.
	0·1953	4·9	

Comparison of the P_2O_5 excretion during the existence of severe melancholic emotional disorder with that obtaining when recovery had taken place, affords some interesting points in the two following cases. The period of mental disturbance is associated with a striking disproportion in the amount of the respective phosphates, which lessens during convalescence until the normal ratio is attained. This character is especially noticeable in the case of the male patient; in which, further, there is seen a significant discharge of glycerinphosphoric

acid, at the time when the disturbance of cerebral function was greatest. Here, again, this disproportion is mainly the effect of a decline in the alkaline phosphates, though there is some decided absolute excess of the earthy phosphates on the first two days, in the case last mentioned.

Each patient received the ordinary diet, throughout. The man, at no time, displayed much energy; the woman was employed at light work in the laundry, when the acute distress subsided.

XVI.—Acute melancholia. J. C., male, 46. Admitted on Oct. 31, 1884, in a state of profound dejection, almost amounting to stupor; had inflicted a dangerous suicidal wound on front of neck a few weeks prior to admission. Stated that the impulse to destroy himself arose suddenly, and he made no effort to resist it. No delusion. Motionless and silent, until roused by other persons. In good bodily condition.

November 1–2*nd*, 1884.

N	P_2O_5	R	Remarks.
5·109	1·309	25·6	
	0·385	7·5	
	0·33	6·4	
November 2–3*rd*.			
8·211	1·197	14·5	
	1·05	12·7	
	0·03	0·3	
November 3–4*th*.			
5·363	0·7597	14·1	
	0·8104	15·1	
	0·0506	0·9	
November 4–5*th*.			
6·375	0·642	10·7	Less depressed.
	1·19	18·6	
	0		
November 5–6*th*.			
	0·534		
	1·036		
	0		
December 6–7*th*.			
9·164	0·8	8·7	Greatly improved: still morbidly quiet and inactive.
	1·0	10 9	
	0		

December 8–9*th*.

N	P_2O_5	R	Remarks.
13·553	0·988	7·2	
	1·146	8·4	
	0		

January 2–3*rd*, 1885.

8·8	0·48	5·4	Recovered.
	1·184	13·4	
	0		

January 4–5*th*.

9·6	0·48	5·0	
	1·2	12·5	
	0		

XVII.—Acute melancholia. E. L., female, 23. Admitted Dec. 6, 1884, showed great melancholic agitation; imagined that some stuff had been given to her, which had dried up her inside; said she "felt right dried up, as though there were not a drop of anything in" her. Feeble and reduced.

December 10–11*th*, 1884.

9·9	0·824	8·3	
	0·944	9·5	
	0		

December 12–13*th*.

8·379	0·46	5·4	
	0·979	11·4	
	0		

December 13–14*th*.

6·021	0·444	7·3	Much improved: calm.
	0·756	12·5	
	0		

January 19–20*th*, 1885.

7·604	0·341	4·4	Quite well.
	0·874	11·4	
	0		

January 20–21*st*.

8·77	0·403	4·5	
	1·2307	14·03	
	0		

Apparently, ether narcosis does not always liberate glycerin-phosphoric acid. In the first case, given below, total suppression of the alkaline phosphates was observed in the urine voided soon after the narcosis; four hours later, the excretion

of those salts had reached an amount much exceeding that present in the urine before the ether was administered.

XVIII.—Ether narcosis, of about 40 minutes' duration, for removal of cystic tumour from breast. The patient, E. O., 71, was a feeble old dement; inactive, but a fair eater, and in good bodily condition.

Feb. 12th, 1885.—The urine, passed just before the narcosis was commenced, contained—

N	P_2O_5	R	Remarks.
0·641	0·032	4·9	
	0·032	4·9	
	0		

Fifteen minutes after end of operation—

0·312	0·026	8·3	
	0·0		
	0·0		

Four hours later—

0·834	0·0475	5·6	Urine has a strong etherous odour.
	0·1128	13·5	
	0		

XIX.—Ether narcosis, of 20 minutes' duration, for amputation of digit. The patient was a fairly robust, middle-aged, woman. Two and a half hours after the operation, the urine had a powerful smell of ether, and contained—

0·0987
0·4794
0·0282

The number of the foregoing analyses, without doubt, is too small to permit of safe generalisation upon the special facts displayed in them. Their value also is diminished by the circumstance that the total renal excretion of several successive 24 hours' periods was not always obtained for investigation. The explanation of that defect lies in the difficulty of collecting the urine of insane persons. Even when the catheter is frequently and regularly employed, the patient occasionally will micturate in bed, and thus frustrate the attempt to secure that 24 hours' excretion. The more intelligent patients are prone to cause failure by simultaneously evacuating the bladder and rectum into the same vessel. To avoid cumbering

literature with false data, which might insidiously originate from such causes of error, I frequently used the urine of somewhat indefinite periods.

Moreover, the question of the composition of the urine serving as an exponent of tissue change proceeding in the nervous system is beset with many conflicting facts and opinions. For instance, glycerinphosphoric acid is a decomposition product of lecithin—a substance which is abundant in nervous tissue, especially the grey matter. Were the occurrence of lecithin co-extensive with only that tissue, we should possess a sure index of some forms of nervous metabolism; but lecithin also exists in blood, bile, pus, milk, and yolk of egg, hence the significance of an excess of glycerinphosphoric acid in the urine is much reduced. Indeed, Lépine[1] at first regarded the presence of that acid in the urine as a diagnostic sign of fatty degeneration of the liver. Since then, he has found it in some cases of grave anæmia, icterus, enteric fever, and acute pneumonia, besides the nervous cases previously mentioned. Zuelzer has met with it in diabetes mellitus and "paralytic dementia," in addition to the instances recorded at the commencement of this paper. One does not learn from the observers mentioned, whether the acid in question were daily excreted during the course of those diseases, or were only casually found.

Dr. Speck, in an elaborate article[2] upon the relations of mental activity to tissue change, gives the results of his investigations concerning the urinary excretion, and the consumption of oxygen, and the elimination of carbonic acid. He summarizes his conclusions as follows: "The final result of the experiments is this, that mental exertion produces no direct effect upon the general tissue change. The molecular processes in the brain, which constitute its basis, consequently, either are not oxidation processes, or they are so slight that they are inaccessible to our methods of research."

However true that may be with regard to physiological conditions, it surely is not legitimate to argue therefrom that pathological events in the brain cannot produce recognisable

[1] See annotation in 'Lancet,' Sept. 23rd, 1882.

[2] 'Archiv für Experiment. Path. u. Pharmacol.,' Dec. 1881, Bd. xv. S. 81.

effects in the urine? Reading Homer, or criticising German and French scientific works (as in Speck's experiments), cannot be supposed to involve retrograde metabolic changes in the brain similar in nature, or equal in degree, to those which find expression in the phenomena of an epileptic fit, or of a non-hæmorrhagic apoplectic seizure?

It would occupy too much space to discuss all Speck's objections[1] to the views enunciated by Zuelzer; one of them, however, is based upon the smallness of the amount of P_2O_5 in the brain, and requires some consideration. Following Bibra's data, Speck estimates that only about 4·1 grammes of P_2O_5 are contained in a human brain, and thus "an extraordinarily intense tissue change must be surmised if it should effect any considerable alteration in the normal daily excretion of about 3 grammes." But, apparently, Bibra and Speck have ignored the lecithin of the brain, which contains 8·798 p.c. P_2O_5 (Hoppe-Seyler), and which, according to Lépine's calculations,[2] raises the total P_2O_5 in a human brain, weighing 1350 grammes, to about 13·5 grammes.

That some of this should, by pathological action in the brain, attain excretion through the kidneys, and effect perceptible alteration in the composition of the urine, can scarcely be doubted. The following quotation from the valuable work by Dr. Cranstoun Charles[3] has an important bearing upon this point:—

"The embryonic brain is much richer in water and poorer in cholesterin, &c., than the developed brain. Further, we find that the lower a mammal stands in the animal scale the richer is its brain in water and the poorer in ethereal extract (chiefly cholesterin and *lecithin*), and accordingly the nearer does it approach to the embryonic brain, whilst the higher the animal's position the poorer is its brain in water, and the richer in the ethereal extract. Some of the constituents removed by the ether are probably compounds chiefly derived from retrograde metamorphosis, and as this is possibly most

[1] They do not relate to the glycerinphosphoric acid—at any rate that body is not mentioned by Speck, and at the date of publication of Speck's article, Zuelzer had only recently made his discoveries respecting it.

[2] Loc. cit., 'Rev. Mens.'

[3] 'Physiological and Pathological Chemistry,' 1884.

active in the highest brains, we can readily understand why the amount of ethereal extract increases in brains of high development. That it is indicative of activity may also be seen from the fact that the very active medulla oblongata and the continuously active spinal cord are poorer in water and very much richer in ethereal extractives than the brain substance itself. In the case of cholesterin, also, it has been shown (Flint) that the blood of the carotid artery is poorer in this body than that of the internal jugular vein."

Now, if these extractives be so intimately associated with the special manifestations of nervous functions, it is reasonable to suppose that their decomposition products must be eliminated when those functions are annulled by a descending metabolism. Cases I. to III. seem to indicate this.

There can be no doubt that all future analyses of the urine, made in connection with nerve states, should include determination of the glycerinphosphoric acid, the alkaline, and the earthy, phosphates—the amount of each being recorded separately.

Clinical Case.

ON SENSORY EPILEPSY. A Case of Basal Cerebral Tumour, affecting the Left Temporo-sphenoidal Lobe, and giving rise to a Paroxysmal Taste-sensation and Dreamy State.

BY JAMES ANDERSON, M.D.,

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G.B., aged 23 years, a schoolmaster, was sent to me, on the 19th of August, 1885, by Mr. Waren Tay, whom he had consulted for loss of vision in his left eye. He told me he had been well till eighteen months previously, when he began to have attacks of a "peculiar sensation" passing down the right arm into the hand, and causing a sensation of swelling in the hand. The feeling, he said, then passed up the spine from the level of the shoulders to the head, spreading over the back part of the "brain" as a "cold sensation," not extending to the front. He frequently shivered when the fit was upon him; but the "peculiar sensation" was confined to the parts mentioned. At first the attacks were rare, once in six months; then they became more frequent, till he had one daily, and sometimes latterly as many as fifteen during the day. During the previous week he had had about four attacks daily. In answer to the question if he knew when the attack was coming on, he replied that he knew about a minute before by "a sensation in his mouth," "a rough bitter sensation," which lasted during the attack. He generally had an attack after any excitement. At first he always saw "a childish scene," meaning a scene of his childhood, the same with every attack, "a trivial scene, not worth remembering," but which always came up till six months before, when the "sensation," *i.e.* the peculiar sensation in the right arm, became more frequent. In this scene he seemed to hear the voices of a woman and children; but the scene and circumstances passed from him, he said, when the fit passed off. He showed some reluctance to talk about the scene; but when I pressed him in a subsequent interview to describe it to me, he said the woman was a neighbour, and that he was there to play with other children, and she was scolding them. The incident had actually occurred, apparently, but there was nothing to impress it upon him—"a trivial little scene, seems absurd that it should recur."

Although when he came to me the scene had ceased to recur, he still heard voices when he had the "peculiar sensation," not speaking to him but in conversation. There were never any chewing movements or smacking of the lips. This I ascertained from his relatives. The attacks, he said, would not have been noticed by those around, unless their attention had been called; they lasted "only a few minutes," recently they had been even shorter. At first he did not know what was going on around, but now he could tell. At a subsequent interview, and in *answer to a leading question*, he said that occasionally at the time of the "peculiar sensation" he had a sensation of smell which he could not define—not strong, "decidedly not pleasant," "did not last long, was not very bad, did not always come, and was not present at first, only for the past four months."

He was a tall, well-built man, somewhat inclined to stoutness, "had made flesh for two years." His features were somewhat thick and blurred, the mouth deviated slightly to the left, and opened more widely at the left when he showed his teeth. He did not fix with the left eye, which diverged slightly outward. He had lived chiefly in the neighbourhood of Boston, Lincolnshire, and for the previous eight months in Dorsetshire; had always been healthy, and had never had any venereal disease. He had been a teacher for ten years, and was evidently a thoroughly intelligent and thoroughly reliable man. His father and a brother died of consumption, the former at 42, the latter at 29 years of age. His mother, two brothers, and two sisters are alive and all fairly well. One sister died of empyema (?), and two others of typhoid fever. (*September* 1886.—Since writing the above I have examined one of the above sisters. She is suffering from rapidly advancing phthisis.)

His left eye was discovered to be defective in 1882, when he was examined by Dr. George Kirkwood, of Peterborough, before admission into St. Peter's College. He could read ordinary newspaper-type with it, however, and he suffered no inconvenience from it until the end of August 1884, a year before he came to me. At this time his eyes "broke down," his left eye pained him, and the sight of it grew steadily worse, till in October he consulted Dr. Kirkwood, who has kindly informed me that he found as follows:—"After the use of homatropine the vision of L. was $\frac{5}{200}$, with a hypermetropia of $\frac{1}{16}$, that of R. was $\frac{20}{200}$ with H. $\frac{1}{16}$. There was marked retinitis and irregularity of the disc in L. and congestion of the disc in R." Dr. Kirkwood applied empl. lyttæ to the temples, and gave him liq. hydrarg. perchlor. with iron. Three weeks later he prescribed spectacles, but with the correcting lenses, V. of L. was only $\frac{20}{200}$, V. of R. $\frac{20}{30}$ nearly. A few weeks afterwards my patient left St. Peter's College, and Dr. Kirkwood did not see him again.

Subsequent to this time the sight of his left eye continued to fail, and during the two months previous to his coming to me it had failed rapidly. In April 1885, *i.e.* four months before he came to me, the sight of the right eye failed rather suddenly in the course

of a week; "spots in front of the music," so that he could not read quickly. *At no time* had there been headache, vomiting or convulsion, and only recently occasional giddiness.

Examination showed that his heart and lungs, his kidneys and other abdominal organs, were to all appearance healthy. The reflexes, superficial and deep, were normal, and the motor power of the limbs was unaffected. His speech was ready and articulation clear. He was right-handed and had always been so. Neither he nor his relatives could tell me anything as to the facial deformity, which resembled imperfect right facial paralysis, but was so slight, that when the face was at rest the only noticeable abnormality was the shallowness of the right naso-labial furrow. There was no apparent loss of sensation in face or limbs. This was carefully tested. Smell was normal with the right nostril, very defective or completely absent with the left. Common sensation good in both nostrils. Taste was apparently somewhat defective on both sides; but his answers were not decisive. The hearing distance for a watch was 5 feet with each ear.

As above noted, there was slight external strabismus of the left eye, noticed in 1882, and possibly present longer. Otherwise the movements of the globes were normal, and there was no ptosis. R. pupil $2\frac{1}{2}$ mm. acted well to light and with accommodation.

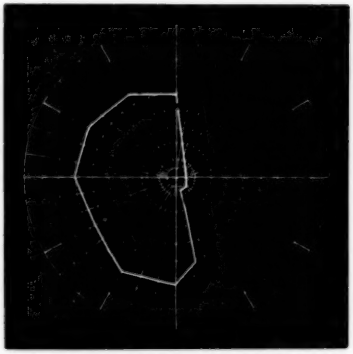

FIG. 1.—Right Visual Field of George B., Aug. 21st, 1885, for white object, 8mm. square.

L. pupil $3\frac{1}{2}$ mm. acted well with accommodation, only consentaneously to light. V. of. L. bare perception of light in temporal part of field, V. of R. $\frac{18}{60}$ partly and J. 1, also with +2 D, the lens he was using. The R. visual field was of full extent on the nasal side, as shown in the accompanying chart, but crossed the vertical through the fixation point by only a narrow margin. The colour vision and nasal half of the colour fields of the R. eye were normal. *Ophth.—L.* Disc dull white, edge sharply defined. Veins of normal size, arteries very slightly diminished. No perivascular

thickening or evidence of past retinitis. *R.* Outer half of disc atrophic, glistening white; inner half still pink, although not so pink as it should be. Vessels normal. No perivascular thickening or exudation.

On August 27th, Dr. Hughlings-Jackson kindly admitted him under his care at the London Hospital. Here the special senses of smell and taste were repeatedly examined, confirming the above results, the slight defect of taste being generally more marked on the left side. He took the bromide and iodide of sodium during his stay in the hospital, which he left on the 19th of October, 1885.

On October 23rd I again examined him, and made the following notes:—Mental condition much deteriorated, extreme hebetude, face expressionless, speech slow and monotonous, could not remember the word "hospital," and although wakening up occasionally into a state of much anxiety regarding his own and his mother's future, he yawned incessantly during the examination. His sister states that he rarely speaks, is generally in a somnolent condition, that he wanders, remembers nothing, and is always strange. He has had no headache, and vomited only once in hospital, from indigestion, he states. The divergence of the mouth to the left is now better marked. There is no definite paralysis or loss of general sensation. There is slight ptosis on both sides, especially the left, but both lids can be raised on request, and there is no ocular paralysis. V. of L. doubtful p l.; of R. $\frac{1}{10}$ and J. 16 with difficulty. R. visual field for a white object differed scarcely at all from that already recorded—temporal hemianopia. The fields for red and green were markedly and regularly contracted, especially the former. *Ophth.—L.* Paper-white atrophy of disc with small arteries and normal-sized veins. *R.* Inner part of disc still pink, but definitely paler than on last examination. Arteries and veins of fairly normal size. No other changes visible in fundi.

From this time till his death I visited him at his home. The mental hebetude gradually increased till his answers became incoherent, yet in flashes his mental state was perfectly clear. A brawny fulness came in the right side of his neck, which varied but did not disappear. He became considerably fatter and more somnolent. Vision got gradually worse, and the nasal half of the right disc got so pale that it could scarcely have been distinguished from perfect atrophy; would certainly have been called so by an observer seeing it for the first time. The arteries, however, remained of full size, in marked contrast to the diminished arteries of the left side.

On December 28th he had a "fit" on getting out of bed, became "stiff all over," and remained unconscious for half an hour. There were no chewing movements and no clonic convulsions. When he became conscious he vomited, and this was repeated frequently till I saw him next day. He apparently knew nothing of what had happened, and lay in a semi-comatose condition. There was no aphasia or evident paralysis. On examining the right disc I thought that its edge was slightly blurred as compared with its previous sharply-cut outline. On January 2nd

there could be no doubt as to the blurring of the edge and fulness of the veins, although the centre of the disc was still white; the left disc remained in its condition of complete paper-white atrophy. On January 17th, after repeated vomiting and giddiness on the 14th, I found the right disc completely obscured by exudation, cloudy white in the centre with a considerable hæmorrhage at its upper edge.

From this time the vomiting recurred at intervals, with occasional attacks of flushing and perspiration followed by pallor. His limbs were stiff, the knee-jerks increased, ankle-clonus was present, and the superficial reflexes were abolished; he passed his evacuations under him. A few more hæmorrhages appeared on the right disc, the reflex from which was now as red as the rest of the fundus; and some glistening white spots appeared near the macula. There was no change on the left disc. Latterly he was probably quite blind; the movements of the globes were less free than previously; but there was no definite ocular paralysis.

On March 4th he passed rather suddenly into a state of complete coma. His left hand was occasionally raised to the left side of his head; the right lay almost perfectly powerless by his side; the knee-jerks were normal, and there was no ankle-clonus. He died on March 6th.

The ocular symptoms in this case pointed definitely to a basal tumour pressing on the left optic nerve in the first place, afterwards involving the left half of chiasma and left optic tract, and finally involving the remaining fibres of the right nerve. I believed also, from my acquaintance with Dr. Hughlings-Jackson's observations, that the paroxysmal taste-sensation and dreamy state, &c., justified the diagnosis that this tumour in some way, *i.e.* directly or through interference with vascular supply, involved the left temporo-sphenoidal lobe, inducing periodic discharges in the region of Ferrier's centre for taste (possibly also in his centre for smell), and in neighbouring highest centres. I use this last term in the sense in which it is used by Dr. Jackson. At this time I happened to mention the case to Mr. Nettleship, who referred me to a case published by him,[1] in which there were similar ocular symptoms. The similarity between the cases was, I found, surprisingly close, not only as to the ocular symptoms, but also in the occurrence of a paroxysmal sensation. Mr. Nettleship thus summarizes his case:—"Failure of left eye (to blindness) passing into atrophy of disc; later, paralysis of left third nerve, and loss of right half of right visual field with evidence of atrophy of disc; discharge of bloody mucus from left nostril, and late appearance of tumour behind left angle of jaw. Death seven years after onset of symptoms; large

[1] 'Ophth. Soc. Trans.,' vol. iv. p. 285.

tumour compressing left optic nerve, chiasma, and tract, and left third nerve." Also it is noted that for several years before death he had been subject at irregular intervals to "fits," which consisted of a sudden feeling of suffocation in the nose and mouth, followed by pain in the front or back of the head, but no loss of consciousness. The similarity in symptoms between these cases was so close, that in writing Dr. Jackson I ventured to say that I believed the tumour would be found in the same position as Mr. Nettleship's, but involving a wider cortical area. The result, as will be seen, fully justified this diagnosis. Not only was the tumour in the same position, but, except in the matter of size, the description of the tumour in Mr. Nettleship's case might be used as the description in mine. Through Mr. Nettleship's kindness I have been able to examine the tumour in his case, and I believe it may be taken as an earlier stage in the development of the large tumour present in my case.

Autopsy (March 7th, 1886).—Body extremely fat, two inches of adipose tissue on the abdomen; skull-cap thick, but readily separable from the dura mater. On the vertex the brain seemed perfectly normal; but on drawing it over to the right it was evident that there was a large tumour at the base. In removing the brain a large amount of ventricular and subarachnoid fluid escaped. Part of the tumour was adherent in the pituitary fossa, and this was removed subsequently. The adhesion of the tumour to the bone was not dense; little more than that between dura mater and skull. In the position of the pituitary fossa was a globular space floored by dense, convoluted, and somewhat roughened bone; its antero-posterior diameter was about 1¼ inches, its transverse about 2 inches, of which last half an inch lay to the right of the middle line, an inch and a half to the left. The part of the tumour remaining in the base of the skull extended slightly into the left orbit along with the structures in the left cavernous sinus and the left optic nerve, which were involved in it; the tumour-growth also passed through the left foramen ovale and foramen rotundum, which were about thrice their usual size. The right optic nerve and structures in the right cavernous sinus were pressed outward by the tumour, but were not directly involved in it.

The tumour is a large mass, consisting of a central soft greyish structure, occupying the position of the interpeduncular space, adherent in the pituitary fossa, and surrounded by masses of steel-blue blood cysts, as shown in the accompanying woodcut taken from a drawing, for which I am indebted to my friend Mr. Jonathan Hutchinson, Jun., and which was made from the brain in its recent state. The cysts extend only half an inch to the right of the middle line, but on the left they extend, large globular masses, backward and outward, undermining the posterior three-fourths of the temporo-sphenoidal lobe, which originally surrounded

the cysts somewhat more than is indicated in the drawing. Anteriorly the tumour extends to the posterior orbital convolutions, and there is evidence of recent inflammation in the longitudinal fissure at this point; posteriorly it extends to the anterior border of the pons, and along its left margin, compressing considerably the left crus. The left olfactory nerve must have been early destroyed; but, owing to the softening and distortion of parts in this region, no part of the cerebral attachments of either nerve can be discovered. The right optic nerve, right half of chiasma and right optic tract lie clear of the tumour, although evidently much

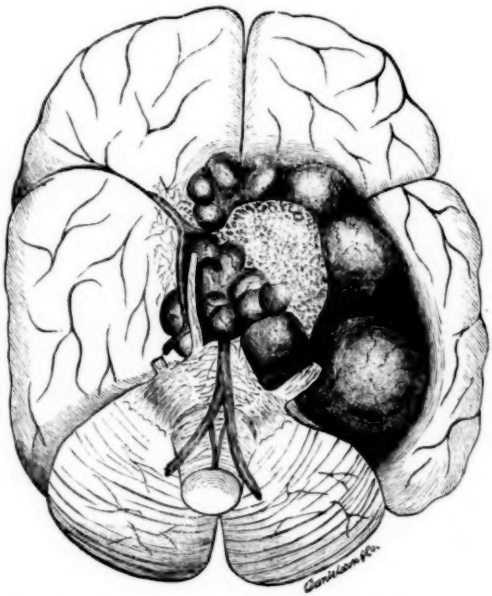

FIG. 2.—Base of Brain, George B., March 7th, 1886, after a drawing by Mr. J. Hutchinson, Jun.

pressed by it. The left nerve and tract are so flattened out as to be almost indistinguishable on the anterior surface of the tumour imbedded among the cysts; the right third nerve lies on the under surface of the tumour, the left probably passes through it; but it is not visible, and was not seen during removal of the brain. The nerves behind this appear quite normal, including the left fourth nerve. The cerebral arteries, so far as they can be traced, are normal in appearance, the left carotid and its branches large, and imbedded among the cysts. The floor of the third

ventricle is thinned to a mere membrane, and the ventricles were evidently greatly dilated; the cerebellum, pons, and medulla are apparently unaffected.

This case is interesting from various points of view especially taken in conjunction with Mr. Nettleship's. Let me emphasise a few of these points.

(1.) The tumour clearly arose from the pituitary body, and was in the first place at least of slow growth. The bone under and around the pituitary fossa was hollowed out, with the formation also of new bone in the neighbourhood. The foramina on the left side of the middle cranial fossa were enlarged, the nerves passing through them being surrounded by prolongations from the tumour, and apparently uninjured. In Mr. Nettleship's case death occurred seven years after the first symptoms. In mine it is impossible to decide as to the precise date of commencement, owing to the left eye being in all probability naturally defective from hypermetropia, but the duration of marked symptoms was less than four years. The central part of the tumour is a soft vascular mass, consisting of heterogeneous round and elongated nucleated cells without stroma. The surrounding cysts contain blood only, very indistinctly laminated, and have walls like those of a large vein. In Mr. Nettleship's case there is the same soft cellular growth in the centre, with the same blood cysts extending backward and to the left, the whole being probably in the recent condition about half the size of this tumour. The soft and cystic character of the tumour and its slow growth explain the escape in my case of the structures in the cavernous grooves which puzzled me before death. In Mr. Nettleship's case the left motor oculi was paralysed. As is generally the case in pituitary tumours, there was extreme development of subcutaneous fat and much mental hebetude, but there was at no time sugar in the urine.

(2.) The late occurrence of neuritis in the right eye was, I must admit, a surprise to me, notwithstanding that I was on the outlook for it. I had watched the disc pass into what would have been called complete atrophy by any one seeing it for the first time, and the eye was probably quite blind like the left. The right retinal vessels were of full or almost full size; but I expected to see them gradually diminish as on the left side. Nine weeks before death, however, there appeared in the right eye a neuro-retinitis, with considerable exudation and hæmorrhages. This occurrence coincided with the onset of vomiting and headache, both previously conspicuous by their absence. Had proof been wanted that this trio of symptoms depends in no way on the size of the cerebral

tumour, and in very little degree at least on proximity of the tumour to the optic nerves, the present case would have amply sufficed.

Was the cause of these symptoms the slight recent inflammation of the meninges just in front of the tumour? Is local meningitis absolutely necessary to cause the trio of symptoms? I fear I cannot add much to what has been done in the way of answering this question. The meningitis in front of the tumour was certainly recent, and there was as certainly very little evidence of inflammation around the tumour at other points. Transverse sections of the optic nerves about a centimetre behind the orbits present, on microscopic examination, characteristic points of agreement and difference. The outer sheath is much alike in both, and, so far as I can see, in no way differs from normal. The inner sheath is more matted in the left, much more intimately blended with the periphery of the nerve, and sends into the nerve much larger trabeculæ. In neither right nor left inner sheath is there any noticeable increase of leucocytes; and I should not have recognised that of the right nerve from a normal sheath. The left nerve, notwithstanding its advanced atrophy, shows throughout fairly numerous axis-cylinders amid granular debris. The fasciculi are much shrunken, separated by thickened vascular trabeculæ, and traversed by a fine reticulum of stained fibres joining at intervals with the processes of branched cells lying among the axis-cylinders and debris—in fact, nervous tissue in a state of typical sclerosis. The right nerve shows but few axis-cylinders, in many parts merely a uniform granular debris, divided by thickened septa, which have no cell infiltration. The fascicular spaces thus marked out contain leucocytes, distinctly more than normal, varying in number in different parts of the section, but not uniformly more numerous at the periphery. At one side of the nerve (I am sorry I cannot say which side) there is an area in which the fasciculi contain branched cells, fine stained fibres passing among the granular material and axis-cylinders, precisely as in the left nerve. The neuritis, which appeared in the right eye nine weeks before death, was evidently of the usual type that occurs in cases of cerebral tumour, simultaneously with headache and vomiting. In ordinary circumstances it would have appeared also in the left eye; but the disc of this eye remained unchanged in its state of sharp-cut paper-white atrophy. Whether the immunity of the left nerve was due to the structural change in the nerve or its meninges, or to the practical disjunction of the nerve from the brain, this case cannot decide; possibly either would have been sufficient. In passing, I may say that

the inflammation of the left fundus in 1884 was, to a thoroughly competent observer, compatible with simple hypermetropic change. It was doubtless, however, a neuro-retinitis of low grade, due to the retro-bulbar pressure, and leaving, as we have seen, little evidence of its presence except the slight opacity of the atrophic disc.

(3.) Lastly, the most important bearing of the case is on the doctrines as to the localisation of cerebral function. In this connection I wish to emphasise that there are two distinct clinical facts to be noted: viz. first, a paroxysmal sensation, and second, a dreamy state. The paroxysmal sensations in Mr. Nettleship's case and mine I consider to be precisely analogous to the motor manifestations of Jacksonian Epilepsy, and to be due to discharges in what Dr. Jackson has termed the middle sensory-centres, just as Jacksonian Epilepsy is due to discharges in the middle motor centres (the cortical part of which is known as Ferrier's motor area). The dreamy state I believe to be due to an extension of the discharge to highest cortical centres, which, as Dr. Jackson says, re-re-represent the body, in the complex combinations of which the reminiscence of my patient is an example. This dreamy state in such a case as this, is indistinguishable from epilepsy proper, or, rather, it is epilepsy proper, consisting, like it, of discharges in the "highest centres." It is analogous in mode of causation to the general convulsions and loss of consciousness, which closes a severe epileptiform seizure, but differs from it in the severity of the discharge, resembling in this latter point the cases of *petit mal*, where the discharge is sufficient only to produce a state resembling reverie.

To return to the paroxysmal taste-sensations which formed the starting-point of my patient's attacks. I have already stated that the tumour in this case arose from the pituitary body, and grew chiefly to the left and backward. I judge of this not only from the tumour itself, but also from Mr. Nettleship's case, which I consider shows an earlier stage in the growth of this tumour. From the position and relations of the pituitary body, it is clear that the first part of the cortex involved by the tumour would be the anterior part of the inner border of the temporo-sphenoidal lobe—in other words, the area in which Ferrier has had grounds for locating the centres of taste and smell, at the tip of the gyrus uncinatus. Producing by pressure an instability in the grey matter of this area, I believe that the tumour was the cause of the paroxysmal taste-sensations which commenced the attack, and that from this point the discharge spread to neighbouring centres.

Dr. Hughlings-Jackson has observed and noted, although not published, numerous cases of paroxysmal sensation, com-

bined with dreamy state; and it was owing to my acquaintance with his views that I was led to investigate the present case in detail. McLane Hamilton has recorded a case of paroxysmal sensation of smell, in which chronic meningeal inflammation over the right uncinate gyrus and sclerosis of subjacent tissue was found *post mortem*. Such cases as these go far to substantiate clinically the accuracy of Ferrier's observation. They are probably more numerous than is generally supposed; this owing partly to the fault of the patient, partly of the physician. The patient may be stupid or careless in observation, and in the expression of what he has observed; or, on the other hand, like my patient, he may be unwilling to speak of it, as being "silly." I need hardly say that in all cases, but more specially with stupid patients, where the temptation is greatest, one must avoid putting leading questions. One such question may destroy altogether the value of subsequent examination. On the side of the physician, every medical man ought nowadays to be aware that the occurrence of these paroxysmal sensations or of the dreamy state is, as Dr. Jackson has shown, an important symptom, foreshadowing grave cerebral disease, as the first signs of tubercular change at the apex of a lung portend coming phthisis. In both cases the disease may be curable; in both it may be beyond cure. In either case it is our duty to know and recognise it from the first, and to guide our patient accordingly.

Critical Digests.

ON THE RECENT ADVANCES IN OUR KNOWLEDGE OF TABES DORSALIS.

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(50) 'British Medical Journal,' April 4, 1885.
(51) 'Path. Trans.,' vol. xxxvi., p. 48.
(52) 'Path. Trans.,' vol. xxxvi., p. 352.
(53) "On Charcot's Joint Disease."—'Lancet,' July, 12, 1884.

54) 'Cases of Joint Disease with Locomotor Ataxy."—'Clin. Soc. Trans.,' vol. xviii.
(55) 'Brit. Med. Jour.,' vol. ii. p. 886.
(56) "Ueber Kniegelenke Affection bei Tabes."—'Deutsche Med. Woch.' Dec. 10, 1885.

THE recent advances in our knowledge of the Tabes Dorsalis, or Locomotor Ataxy, are in the direction the former advances have been in other diseases. Thus many years ago a doctor was content with the diagnosis of jaundice, as are the laity now; but at the present time the physician who did not diagnose the cause of which jaundice is only a symptom, would be looked upon as ignorant. In the same manner it is to be hoped that we are within measurable distance of the time when the diagnosis of a mere symptom, such as ataxy, will not do, but instead, we shall be able, from the grouping of symptoms, to say exactly what parts are affected. To a certain extent we are able to do so now; thus Pick (1) points out that we may have a cortical ataxy, a cerebellar ataxy, a spinal ataxy, and a neuritic ataxy. Still the phrase locomotor ataxy is usually reserved for the spinal form, and future investigation will be directed to discovering the exact parts of the spinal cord affected, and the reason why the patient should be ataxic when those parts are affected. Erb (2) has well pleaded for a more careful consideration of the facts of each case before adopting any theory, either Leyden's sensory one, Friedreich's motor one, or Strümpell's modification of the two. He throws out the suggestion that the ataxy must be due to some affection of that part of the motor tract, between the centres of will and the anterior cornua cells, which has to do with the co-ordination of movements. As disproving the sensory theory, he contrasts two cases, both of which were highly ataxic; but in one there was great sensory perversion, in the other none at all.

The best recent paper on locomotor ataxy, looked at from the point of view we are considering, is by Spitzka, (3) who says that the initial lesion is either in the posterior grey horn, or in that part of the column of Burdach traversed by the inner division of the posterior roots as they reach the level of entry into the grey substance. It is distinct from the secondary degenerations which accompany it. The affections of the columns of Goll and Clarke and the cerebellar tract are

truly systemic; that of the middle area of the column of Burdach probably is not, whilst that of the root zones (*bandelettes externes*) certainly is not. The reasons for looking upon the degeneration of the column of Goll in tabes as a true secondary degeneration are, that when the lesion in the primary field is the lower lumbar or sacral, the degeneration in the column of Goll is limited to its postero-internal lower; when the primary disease is lower dorsal or upper lumbar, the rest of Goll's column is affected; and when the brachial, the comma-shaped area of Burdach's column just external to Goll's column, is affected; the degree of the affection of these areas bears a direct relation to the degree of motor ataxy. Thus, when there is motor ataxia of the arms, the comma-shaped area of Burdach is involved; when of all parts of the lower extremities, the column of Goll. Both the column of Goll and the direct cerebellar tract are under the trophic dominion of the posterior roots; thus the amount of degeneration in them generally corresponds, and hence static ataxy is found in advanced cases of tabes. Not only does the degree of the involvement of the column of Goll vary with the amount of motor ataxy, but it is unconnected with any other symptom, and affection of the cerebellar tract is connected with no other symptoms than static ataxy, and that of the column of Clarke is also connected with this symptom. It appears from several carefully recorded cases, that there is a small area with its base at the periphery of the cord, one side along the inner border of the posterior root for a short distance, and the remaining side joining the other two, which is affected in cases in which there are pains and hyperæsthesia, and profoundly diseased when there is analgesia, and in no case in which there was pain-disturbance was this area healthy. The affection of the bandelettes externes causes the abolition of the tendon reflexes. The extreme anterior part of Burdach's column is hardly ever diseased, and we have not at present been able to localise the lesion of disturbances in the tactile sense, or of sexual, bladder, rectal, or trophic troubles. The destruction of cortical control is inimical to the development of typical ataxy; hence patients who have early locomotor ataxy, and then develop cerebral symptoms, lose their ataxy.

Bechterew, (4) as a result of his observations on the development of the cord in the fœtus, comes to the conclusion that there are at least two distinct areas in Burdach's column, viz. one which stretches from the posterior root entry, along the periphery of the cord (it would seem probable that this corresponds with the analgesic tract mentioned above), and an anterior which lies along the inner border of the posterior cornua behind the posterior commissure.

Krauss, (5) as far as he goes, agrees with Strümpell in the position of the primary lesion, and the secondary lesions of Goll's column, as quoted above in Spitzka's article, which is founded partly on Strümpell's results. But here it may perhaps be pointed out that Spitzka totally disagrees with many of Strümpell's localisation of symptoms; for example, the latter thinks that the bladder-disturbance is due to disease of Goll's columns; but apparently this is totally incorrect, not only pathologically, but from a comparative anatomy point of view, for the porpoise, in which Goll's columns are very small, has no posterior extremities, although the bladder is developed as in other mammals. Krauss himself does not offer any views on the position of the symptoms in connection with the lesions, but records ten cases, to show that there may be ataxy without any change in grey matter, and that the primary seat in the root zone may be affected without any patellar reflex alteration, both of which statements are easily understandable.

As also tending in the direction of limiting more exactly the parts of the cord affected may be mentioned a short paper by Lissauer, (6) who describes an ascending bundle of fine fibres which are first on the inner side, later in the posterior horn. This bundle, he says, is diseased in locomotor ataxy.

Westphal (7) gives a very full record of a case. In the cervical region the inner part of the column of Goll and the boundary line between it and Burdach are affected, lower in the cord the latter gets nearer and nearer the posterior cornua. In the upper dorsal region it is triangular, whilst the Goll part is broader. In the mid-dorsal region both tracts are larger. In the lower dorsal and lumbar regions there is affection of the apex of the post-median fissure. In the cervical region the lateral column is slightly affected, in the lumbar regions the anterior

root zones, and in the medulla oblongata the cuneate and slender fasciculi. Peripheral neuritis of the right lower extremity, and also changes in the roots of the spinal nerves, are present. Westphal attributes the non-abolition of the knee-phenomena to the root zone in the dorsal and lumbar regions remaining free. There was absence of ataxy, which he thinks remarkable, and thinks that probably the cause of this anomaly is to be sought in the intensity of the degenerative process. The extreme disturbance of sensation which was present was due to the peripheral neuritis.

Putzel (8) describes the histological appearances of a case; but from his description we cannot draw any conclusions as to the relation of symptoms to the disease of distinct strands, for all he says is that the extreme anterior part of the posterior columns was not affected (in this point he agrees with all other observers), the grey matter was normal, the change was least marked in the posterior root zones, and the posterior parts of the posterior columns; but there was a general increase of neuroglia in many parts of the cord. Post-mortem examinations on cases of tabes are few, and careful descriptions of the cord difficult to make; still it will be seen that all the recent literature goes to show that the posterior columns and posterior cornua are a very complex part of the cord, and contain many tracts. Only the most careful observation can show what symptoms are connected with what lesions, whether the various strands are affected by Wallerian degeneration secondary to affection of a primary field, or whether the disease is a diffuse sclerosis, attacking irregularly sometimes one and sometimes another band of nervous tissue in the posterior columns. The fact seeming to point to the last conclusion is the well-known one that the disease is very likely to occur before, and be followed by general paralysis of the insane (Savage, Insanity, and allied Neuroses), which is well known to be a diffuse disease. Baillarger (9) has recently published two interesting cases showing the association of the two diseases. Turner has published a case (10) in which the posterior columns, lateral columns, and grey matter were affected; and Ormerod (11) has brought up the knowledge of the association of disease of the lateral columns, with that of

the posterior, to recent date. In all probability it will be found that the name locomotor ataxy, as the name of the disease, will have to be got rid of altogether; whilst the name tabes dorsalis might be retained for those cases in which there is primary lesion of the posterior cornua, with secondary Wallerian degenerations; whilst a whole mass of cases, some of which are peripheral neuritis, some are diffuse myelitis, and others a chronic meningitis, will have to be relegated to their respective positions.

The next pathological point which has been hotly discussed is whether the change is a primary neuritis, primarily a sclerosis of the interstitial tissue, or primarily a disease of the blood-vessels. Adamkiewicz, (12) as is well known, brought forward prominently the view that tabes was always due to primary sclerosis of the connective tissue. This was undoubtedly too sweeping an assertion. Schultze (13) has criticised his conclusions; a controversy sprang up between the two authors, and may be found in subsequent numbers of the same periodical. Still it is extremely probable that in many cases it is a primary sclerosis of the interstitial tissue, or perhaps in others a sclerosis due to a meningitis. As an example of this causation may be mentioned a paper by Déjérine (14). Here he records cases in which there was a posterior and lateral leptomeningitis, with a sclerosis of Burdach's and Goll's columns, and also a slight affection of the external part of the lateral columns. Krauss, in the article already quoted, does not incline to the view of a primary leptomeningitis. Ribail (15) records some cases of old people, in some of whom there was a sclerosis of the cord in connection with chronic meningitis, and bony plates in the membrane in the others; but whilst it is possible that the meningitis may have had something to do with the sclerosis, I think there is no doubt that the presence of the bony plates was only a coincidence, for they are so commonly observed without any affection of the cord. As far as I know, no very recent important work has been done with a view of determining which is really the more important factor—a parenchymatous or an interstitial neuritis; but several papers have appeared, discussing the probability of disease of the vessels being the primary lesion. Of these I think the best is

one by Buzzard, (16) in whose case there was much inflammation of outer coat of the vessels of the cord, causing dilatation of their channels. The lesion was widely diffused. The posterior cornu, the cerebellar and crossed pyramidal tracts and anterior cornual cells were affected. The membranes of the cord were notably thick and adherent. Buzzard thinks that here the periarteritis was the primary condition. Demange (17) has recorded cases of the most marked kind in the spinal vessels of the old people, which, he says, were exactly like the changes seen in the cerebral vessels in old people, and which probably, through the medium of miliary aneurysms, lead to hæmorrhages. These changes, he thinks, lead to sclerosis of the cord, but Krauss (*op. cit.*) tested the question by seeing if there was any difference in the vessels in tabetic cases and in cases of secondary degeneration, and he could not find enough to warrant the conclusion that the changes in the vessels were primary in tabes. Demange, (18) soon after his last article, published another case in which the patient, 75 years old, had progressive contraction of the limbs, diffuse sclerosis of the lateral columns, generalised atheroma, periarteritis of the spinal vessels, granular kidneys, atheroma of the cerebral arteries, but no cerebral softening. The sclerosis affected slightly the columns of Goll and the anterior cornua, as well as the lateral columns. The author proceeds at great length to discuss all his cases, and concludes that atheroma of the spinal arteries will lead to sclerosis of the lateral columns, with spasmodic tabes, just as atheroma of the renal artery will lead to interstitial nephritis. But against his reasoning we may point out firstly, that it is by no means universally allowed that atheroma of the renal artery will lead to interstitial nephritis; secondly, that atheroma of the cerebral arteries does not cause sclerosis of the brain, but softening or cerebral hæmorrhage; thirdly, that it is extremely unlikely that atheroma of the vessels would give rise to a system lesion such as lateral sclerosis, for that seems to have been the predominating lesion, although the author calls his cases at the end of his article, ones of diffuse and disseminated sclerosis.

This is a good opportunity for pointing out that it has lately been urged that cardiac disease has some connection with

ataxy. Grasset (19) refers to a short paper by O. Berger and O. Rosenbach (20). The only lesion observed by those authors was aortic disease. Grasset then records two cases that he himself has observed, and collects together a number from other authors. He concludes that valvular lesions other than aortic may exist, and that frequently the cause of the heart-disease is very obscure. He does not consider that the spinal cord and cardiac lesions stand in the relation of cause and effect to each other, but points out that the tabetic cases in which cardiac lesions are present usually have very severe pains, and suggests that, as it is well known that severe peripheral stimulation will affect the heart reflexly, it is possible that the cardiac disease may be caused in this way. I need hardly point out the extreme improbability of this suggestion; since the publication of Grasset's paper no other facts have been put forward to support it. Richardière (21) has published a case of tabes in which, with very peculiar trophic changes, aortic disease occurred. He is surprised that he cannot find any cause for the atheroma which caused the aortic disease, although he mentions the presence of syphilis in his patient. From reading the case through, one cannot help thinking that there is no need for him to draw attention to the fact of the association of aortic disease and tabes, considering that syphilis is assigned as a cause for both tabes and atheroma. We may, I think, conclude this part of the history of tabes with the pretty certain assurance that at present there is nothing to show more than an accidental connection between that malady and heart disease.

There has been much controversy as to the ætiology of tabes; and as is always the case when the exact limitation of a disease is vague, the opinions as to the causation are various. Until we can be sure of the exact pathological lesion in each of the cases, and all authors are agreed as to the pathological lesion which shall be called tabes, it is hopeless to attempt any certain knowledge as to causation; or at least the group of clinical symptoms to which the name tabes is given must be more exact than now—as exact, for example, as the group of symptoms constituting chorea; which, although it has no pathological basis, yet has a distinct ætiology.

To take an example; it is quite clear that all cases of peripheral neuritis must be excluded, and there can be no doubt that many cases that were formerly put down as locomotor ataxy, or tabes dorsalis, were in reality cases of peripheral neuritis. Perhaps the most instructive paper on this aspect of tabes is one by Dr. G. M. Hammond (22). He maintained that the disease could recover, but that the cases which did so were examples of posterior spinal congestion. In passing, one may remark that this at present is the purest hypothesis. In the discussion which followed on this paper, it was pointed out by several speakers that it was extremely probable that the cured cases were not posterior spinal sclerosis at all, but were other diseases, which at present it is very difficult to separate off during life from that malady. Thus Dr. Bartholow mentioned cases of mercurial ataxic and syphilitic gummata of the spinal cord which had been cured. Dr. Webber referred to a case of Déjérine's, in which during life locomotor ataxy was diagnosed, but the post-mortem showed peripheral neuritis. Now had this case been a recoverable peripheral neuritis, it would have been set down as locomotor ataxy recovered. One must also remember that syphilis and hysteria may cause symptoms which would very much resemble locomotor ataxy. Saturnine ataxy is also known; it is curable, but is probably a peripheral neuritis. Post-diphtheritic disorders of a rare nature might easily be mistaken for tabes dorsalis, as might the diseases kak-ke and beri-beri.

As an example of post-diphtheritic ataxy I may refer to a case by Berwald (23). The patient, three weeks after diphtheria, had paralysis of the palate, and soon after pains, deafness, difficulty of walking and using the hands. The author gives references to other cases, and, from an analysis of the symptoms in this one, comes to the conclusion that it is not a peripheral neuritis.

Then again there is Hughes Bennett's case (24), in which there was no disease of the posterior columns, but a number of tumours on the posterior roots, and sarcomatous softening of the medulla, and yet the case was diagnosed during life by every one who saw it as one of locomotor ataxy. Dr. Bennett has carefully considered the cause of the absence of medullary

symptoms, and hence I do not think that Spitzka's (*op. cit.* p. 395) criticism is valid.

A case of saturnine ataxy will be found recorded at the same meeting of the American Neurological Association as that at which Dr. Hammond's paper was read.

Then also there is the question whether in papers dealing with the ætiology of tabes, cases have been excluded in which the tabes was either accompanied by, or secondary to, other diseases of the nervous system. I have already referred to the fact that it often precedes or follows general paralysis, which is well-known to be due to syphilis. Some marked cases showing this association have been recorded by Seguin (25) and by Fournier (26), and Camuset (27) has recorded a case in which without ataxy there was primary sclerosis of the columns of Goll in an old person who was mentally weak.

In the number of 'BRAIN' for January, 1886, will be found abstracted a case by Sakaky, in which peripheral neuritis existed in association with locomotor ataxy, and it is pointed out that several other such cases have been recorded; for example, Krauss (*op. cit.*) mentions some. All these references show the difficulty of separating tabes exactly from various diseases of the nervous system, and hence of discovering its cause.

Belugon (28) discusses at some length, and by the help of elaborate tables, the cause. With regard to syphilis, he points out that the existence of the two diseases is often only a coincidence, because anti-syphilitic treatment did no good to the tabetic symptoms. He concludes that syphilis rarely causes the disease, but that it puts the patient in such a condition that he is very likely to be affected by tabes. Hereditary influence is of some ætiological value, but not of so much as syphilis. Rheumatism is of all causes by far the most important. He is not able to decide upon the value of excesses as a cause, but considers that sexual excess may be one. In a subsequent number of 'Le Progrès Médical' there is a letter from MM. Landouzy and Ballet, to point out that they have also recognised the importance of rheumatism.

It is well known that some authors have pointed out the importance of syphilis in tabes. Erb (29) brings forward 100

cases of tabes, 91 of which had syphilis. The percentage of syphilis among non-tabetic cases at the same clinic being 22·75. Also he says it is noteworthy that tabes usually occurs late in life among those who have acquired syphilis late. Again, in both diseases the proportion of men to women is ten to one, and both are common in the lower classes, rare in the upper. Since the publication of that paper, Eulenburg (30) and Berger (31) have pointed out the strong association between the two diseases. Eulenburg's percentage is not quite so high as Erb's. Berger gives some interesting cases; the most striking is that of a man who, at the age of 68, acquired syphilis, and at 72 years old developed tabes. The percentage in this paper is pretty much the same as that in Eulenburg's. The hereditary influence is well shown in some cases published by Everett Smith (32), who gives an account of a family of thirteen children, of whom five had ataxy, and the disease was also present in the father. It will be seen that at present, syphilis, excesses, hereditary influence and rheumatism, seem to be of some ætiological value, but how much cannot be determined till tabes is more exactly defined.

From time to time rare symptoms are recorded as occurring in the course of tabes. In fact they are often so rare, that one cannot help thinking that perhaps their presence is a mere coincidence. A few of these more unusual accompaniments are here recorded.

Birdsall (33) gives an account of some of the ocular affections. It appears that visual acuteness is nearly always unequal in the two eyes, but patients retain longer the power of seeing near than far. Unusually strong electric currents are required to produce a sensation of light. Hyperæsthesia and anæsthesia may be observed over the distribution of the 5th nerve, the former being accompanied by excavation of the optic papilli. There may be colour-blindness.

G. H. Roger (34) says that out of 32 cases he examined, 14 had diarrhœa, and in 5 of them there was reason to think it had special connection with the disease. It may or may not be accompanied by abdominal pain. It is often sudden, and the evacuations are watery.

Many of the symptoms recently recorded have been of a

trophic nature. Richardière (35) has recorded a case of disease of the metacarpo-phalangeal joint, with luxation of the thumb in locomotor ataxy. In the same case the teeth fell out. The patient thus describes this occurrence. A year before admission into the hospital, the eyes and face swelled, and one by one the teeth fell out without any caries; after this a few pieces of bone fell out from the palate. On admission to the hospital it was discovered that the gums were healthy, that the upper teeth had gone, but that the lower were all there.

Hoffmann (36) records three cases of tabes. In the second, without any cause, the patient, a man of 48, lost in about ten days all the teeth of the upper jaw. Two years after, the tabetic symptoms began. The first case, that of a man aged 59, is peculiar from the fact that a subcutaneous rupture of the tendo Achillis took place spontaneously. This is an excessively rare trophic lesion in tabes. Dr. Lewis (37) and other authors have also described cases in which the teeth fell out.

Unusual trophic changes in the form of grey hair and pigmentary changes over the 5th nerve distribution will be found in a paper by Klinkert (38).

Oppenheim (39) has published a very interesting account of laryngeal crises, and after referring to the fact that sometimes the vagus or recurrent laryngeal has been found affected atrophically, and that it would appear that the symptom may be due to disease of the 4th ventricle, he records a case of a man who had peculiar crises accompanied by dyspnœa. On admission he had a falsetto voice. On deep inspiration there were present much difficulty of breathing and stridor, paralysis of both crico-arytænoidei postici and the right lateral crico-arytænoid, difficulty of swallowing, pain on pressure at the inner border of the sterno-mastoid, and temporary failure of pulse with attacks of giddiness. Then the author gives an account of a patient with very similar symptoms, in whom was found degeneration in the vagal trunk and the recurrent laryngeal. The same author (40) points out the frequency with which hemicrania is associated with tabes. Out of 85 cases he found that 12 had had typical migraine, and of these 12, ten were men and two were women.

Vulpian (41) describes a case which progressed slowly, and was verified by a post-mortem examination. The patient in 1878 was seized with violent epigastric pain, diarrhœa, and vomiting; from this he recovered and remained well two years, when he had a similar attack. He went in and out of the hospital several times, but it was not till 1882 he was finally admitted, when tabes dorsalis, with gastric crises and angina pectoris, was diagnosed. He had repeated attacks of cardiac pain, the association of which with tabes forms the chief interest of the case; he died at last of phthisis.

It is well known that, putting aside cases of Friedreich's disease, cases of ataxy are not common in young children; but Remak (42) gives three cases of various tabetic symptoms in children who had had congenital syphilis and had optic atrophy, one was 12, another 14, and another 16 years old. They were no relations to one another.

Ballet (43) has pointed out that hemiatrophy of the tongue is not very infrequent. It may occur on either side; the affected side is thrown into wrinkles and furrows. There is sometimes a slight contraction with vermicular movements; it points to the affected side; it is of a very early symptom, and produces no effects on deglutition, mastication, or speech. Since this paper, other cases have been recorded.

In England, discussion about Charcot's disease has waxed very warm during the last two years, and although every one has had an opportunity of expressing his opinion, it is doubtful whether our exact knowledge of the subject has been much advanced. If this be a genuine symptom or complication of tabes, it labours under the disadvantage that probably many of the cases in which it exists have not been tabes at all, but peripheral neuritis, or some other disease closely resembling tabes.

In November 1883 I showed (44) a pelvis which, although no history could be obtained, for it was a dissecting-room specimen, was so like to those specimens described by Charcot as characteristic of tabes, that I got my friend Prof. Féré to show it to Prof. Charcot, who pronounced it to be an example of the disease named after him. It certainly is the most

unusual pelvis I have ever seen. It weighed but 7 oz. The bones were extremely thin, this being best shown in the acetabula, which have been pressed in to form two great bosses on the interior of the pelvis. Owing to this deepening of the acetabular cavity, the lesser trochanter had worn away a place for itself at the margin, and thus the cavity was much larger than natural. At the margin of the cavity there was the merest trace of new bone. The specimen is in the Guy's Hospital museum. At the conclusion of my paper, my doubts are expressed whether Charcot's disease is a distinct pathological entity.

Mr. Arbuthnot Lane (45) pointed out that the joints affected in rheumatic arthritis are also affected in Charcot's disease; that the absence of osteophytic growth is not a point of distinction; and finally, Mr. Lane could not find any points of distinction between the two diseases, and considers that when rheumatic arthritis occurs in old people it presents such degenerative changes as to be indistinguishable from Charcot's disease, which, when it is found in strong healthy people, is indistinguishable from rheumatic arthritis. Thus they are one and the same disease, modified by the patient. The same author (46) mentioned a pelvis deformed in a very similar manner to others previously described, and again emphasized his opinion that Charcot's disease is rheumatoid arthritis, modified by the degenerated condition of the tissues involved. Mr. Clement Lucas (47) gave the case of an ataxic man in whom the elbow presented many bony prominences and some crackling; the right foot was swollen, but neither tender nor œdematous, although painful; but Mr. Lane (*op. cit.*) thought this case was one of rheumatic arthritis, and I must say that, on examining the patient, I thought so too. Dr. Buzzard showed (48) a case of Charcot's disease in the shoulder, and it certainly corresponded much more closely to Charcot's description than did Mr. Lucas's case. Cases of perforating ulcers of the foot in locomotor ataxy have been recorded by Dr. Duckworth (49) and by Dr. Suckling (50). Dr. Paddison (51) mentions a case of Charcot's disease. Examination of the spinal cord showed affection of the columns of Goll in the crural region and the posterior columns in the lumbar, and Mr. Hopkins (52)

describes two cases of arthritis occurring in locomotor ataxy. I collected (53) the evidence for and against regarding this disease as a distinct affection, and pointed out that Mr. Hulke, Dr. Moxon and Dr. Goodhart had in discussions at the societies all expressed themselves in favour of the view that it was a modification of rheumatoid arthritis. I enumerated three views which might be held: first, that the joint-disease is a distinct specific arthropathy peculiar to locomotor ataxy; against this it may be said that although the anterior cornua, the medulla and the peripheral nerves have all been said to be the cause of the arthropathy, yet it is highly probable that none of them can be looked upon as constant causes; then with regard to the clinical symptoms, a large number of observers know nothing of the most characteristic symptoms described by Charcot, but recognise the cases as examples of rheumatic arthritis, and many of Charcot's symptoms are also symptoms of rheumatic arthritis; out of six cases of joint-trouble with locomotor ataxy I have seen, there was not one which all observers agreed was rheumatic arthritis, nor one which they all agreed was Charcot's disease. The morbid anatomy of the English specimens is discussed, and also Charcot's account of the morbid anatomy of his, and it is shown that there is no point distinctive of the two diseases; and the case is mentioned of a man whose larger joints presented all the rheumatoid arthritis changes, whilst the temporo-maxillary articulation was exactly like Charcot's description. The second view is that the joint-disease of locomotor ataxy is really the same thing as the joint lesions produced by injuries to nerves, or due to cerebral hæmorrhage. It is quite possible that some of them may be of this nature; but if so, why not at once put them in the same class? References to the literature of nervous arthropathies are given. The third view is that it is really an ordinary arthritis occurring independently of tabes, but modified by the conditions which it enforces. Many arguments, such as that of the lowered vitality, the prolonged rest in bed, the liability to traumatism, are brought forward for this view, and it is urged that no one has ever shown that ataxics are more liable to joint-disease than other people, which they would be if there were a specific arthropathy, for

then they would be liable to that in addition to their liability to rheumatic arthritis.

In Mr. Morrant Baker's (54) paper the first case is that of an ataxic woman, in whom there was flail-like movement of hips and elbow, with bony bosses and grating in the latter; the right metatarso-phalangeal great-toe joint was also affected. In the second case the right knee was affected, and there was some dead bone in the right toe. Post-mortem examination showed sclerosis of the columns of Goll and much increase of bone in the neighbourhood of the knee. In the third case the knees were affected, and both during life and at the post-mortem showed most of the changes described by Charcot, and also some points of resemblance to rheumatoid arthritis. Mr. Morrant Baker came to the conclusion that it was "only in an exaggerated form what had long been familiar to us under the name chronic rheumatic arthritis." He thought the association of tabes and joint-disease was not a mere coincidence, and after rejecting the neurotic view of chronic rheumatic arthritis, he went on to say that it seemed to him "conceivable that the ataxy and joint-disease might be due to some pre-existing depraved condition common to both of them," and in proof of that view he refers to a well-known letter by Dr. Wilks (55), which points out that both pathologically and clinically at present we have no definite disease tabes dorsalis. In the discussion which followed the reading of Mr. Baker's paper, Mr. Barker showed a specimen of the disease. Dr. Dyce Duckworth thought Charcot's position proved; Dr. Hale White considered most of the cases were modified forms of rheumatoid arthritis; whilst Dr. Buzzard and Mr. Barwell agreed with Dr. Duckworth. Sir James Paget thought that in a certain sense it must be looked upon as a new disease, for in the collections of the older observers no specimens could be found. The fact that we named it after Charcot showed we knew very little about it pathologically. "It might well be a rheumatic arthritis modified by its coincidence with disease of the spinal marrow." The extremes were distinct; but as they ran one into the other at the borderland, it might justly be suspected that the diseased joints were really examples of chronic rheumatic arthritis. The next speakers, viz. Dr. Ord, Prof. Humphry, and Mr.

Hutchinson, in the main agreed with Sir James Paget. Mr. Halke criticised Mr. Barwell severely, and felt quite unable to agree with those who looked upon Charcot's disease as a distinct thing from rheumatoid arthritis; in this view Dr. Moxon also most fully agreed. Mr. Morris showed two interesting cases; one of disease of the knee, and another of disease of the ankle, which could not be distinguished from Charcot's disease. The patients were without ataxy. In the second case there was elephantiasis of the leg and perforating ulcer, together with the nerve changes usually found in that disease. Mr. Page, Mr. Macnamara, and Mr. Clement Lucas believed in Charcot's disease as a distinct disease, whilst Dr. Pye Smith did not. Many other speakers spoke; but enough have been referred to show that on the whole the opinion was distinctly in favour of Charcot's disease being a modification of rheumatoid arthritis. Mr. Baker, in his reply, reiterated this view. A letter was read from Prof. Charcot, who expressed his surprise that this was the prevailing opinion in England.

Leyden (56) is very undecided as to the relation of tabetic joint-affections to rheumatic arthritis. On the other hand, at a meeting of the Philadelphia Neurological Society, Jan. 26th, 1885, which was reported in a subsequent number of the 'Journal of Mental and Nervous Diseases,' Drs. Wood, Roberts, and others, read papers on Charcot's disease; the prevailing impression was that it is a distinct disease.

From this review of our knowledge of tabes it would appear that we have not advanced much, but in a way we have, towards the time when one or many distinct diseases with distinct pathologies and distinct symptoms will crystallize out of the mass of conditions now grouped together under the name tabes dorsalis.

ON THE ETIOLOGY OF TABES DORSALIS.

BY JAMES PIETERSEN.

Under the title of "Over de Aetiologie der Tabes Dorsalis," Dr. B. H. Stephan contributes to the 'Nederlandsch Tijdschrift voor Geneeskunde' an interesting paper, summing up all we know from Continental writers on the subject, and raising the interesting question whether Locomotory Ataxy cannot be regarded rather as an affection of the whole cerebro-spinal system, due directly to change in the vessels supplying the nervous system, than as has heretofore been done, attributing all the symptoms to one or more definite cord lesions; and among the causes which favour changes in the blood-vessels he gives syphilis the first place, making it thus indirectly the main, though not the exclusive cause of tabes. Regarding sexual excess as a primary cause, he says, "One can well understand that sexual excess, even as a severe attack of typhus, syphilis, alcoholism, rapidly following and more or less continuous lactation, may now and then favour the development of this affection; but having regard to the relative frequency with which sexual excess is met with, and the relative infrequency of occurrence of tabes dorsalis, it seems to me that the assumption of such as a primary cause is wholly unwarrantable." Exposure to cold, as well as mental and physical strain, he regards, with hereditary predisposition, as of secondary moment. He then quotes Fournier as the earliest promulgator of the syphilitic origin of tabes, whose disciple Erb went so far as to affirm that no one could evince ataxic symptoms without being able to account for a previous syphilitic affection. "The main arguments brought forward by those who deny the syphilitic origin of locomotor ataxy are to be classed under three heads.

"1. The almost inappreciable success of specific treatment.

"2. That the lesions in tabes, limited as they are to certain anatomical tracts, in no way correspond with other syphilitic affections of the nervous system.

"3. The unreliability of the statistics brought forward."

Putting on one side the first two opposing arguments, which he deals with more fully in the body of his paper, he holds that statistics are of great value in the consideration of this question, and that there are three points for us to consider when we look at it statistically.

"(1.) Are the numerical results in the various tables writers have drawn up to show the connection between syphilis and tabes proportionately alike?

"(2.) Do investigators regard the early manifestation of a chancre of doubtful character, or a soft sore, as sufficient warranty that

when tabes occurs a syphilitic cause should be assigned to the malady? [for the distinction between the hard chancre and soft sore, in that they are each due to a separate and distinct virus, is by no means universal].

"(3.) Is the tabulation of such cases where syphilis preceded tabes satisfactory, when a doubtful chancre or a soft sore was considered sufficient evidence to assume syphilis?"

Notwithstanding these weak points, he holds that a remarkable relation exists between tabes dorsalis and earlier syphilis, "for when Bernhardt computes that of 125 ataxic patients, 46·8 per cent. had some syphilitic antecedents, while of 155 others, not ataxic, only 15·8 per cent. had had syphilis (in both cases soft chancre being regarded as non-syphilitic), then undoubtedly some connection must exist between these two affections." He goes further, quoting *Landsberg*,[1] who published a case of locomotor ataxy with precursory syphilis, where the nerve affection yielded to a specific treatment, mainly inunction, and where the malady at the time of publication had almost wholly disappeared, the most undoubted ataxy having manifested itself previously: *Berger*,[2] who relates the history and post-mortem appearances of a case that came under his observation, where syphilis was contracted in the 69th year, followed by slight secondary symptoms. Three years later double interstitial orchitis, with typical tabes, manifested themselves; the diagnosis was verified by the autopsy: *Voigt*,[3] too, draws attention to the access of tabes in old persons where syphilitic infection had taken place in the later years of life.

He does not, however, agree with Erb in giving syphilis as the exclusive cause of tabes dorsalis, for *Eulenburg*[4] could only prove precursory syphilis in 36·8 per cent. of his cases, Berger only in 43·8 per cent. "There are therefore," he continues, "in the development of this spinal affection other causes also at work. What these causes can be, and what the nature of the connection is between syphilis and these causes on the one hand, and tabes on the other, we must follow more closely."

He first of all draws attention to the action of poisons on the system, quoting Strümpell, that observation of such action often is of etiological value as regards cord affections. "We know that certain poisons act only on certain portions of the spinal cord, disturbing certain physiological centres without in any way affecting the whole cerebro-spinal system (instance the action of *strychnine* on the reflex centre: *curari* on the motor end-plates of peripheral nerves: *morphine* on the sensory central organs: *atropine* on the vasomotor and secretory centres). The action of certain poisons, too, is not alone the cause of functional, but also sometimes of anatomical change (e.g. *Plumbism* causing affection of the motor portion of a certain nerve: *Ergotism* evincing not only a clinical but also a pathological likeness to tabes)."

[1] 'Berl. Klin. Wochenschrift,' 1885, No. 33.
[2] 'Deutsch. Med. Wochenschrift,' 1885, Nos. 1 and 2.
[3] 'Centralblatt für Nervenheilkunde,' 1885, No. 8.
[4] Virchow's 'Archiv,' Bd. 99, 1.

"More and more probable does it then appear," he continues, "that infectious vessel-affection (one species of poison-influence) plays etiologically a more important part in lesions of the spinal cord than up to the present it has been accredited with. Various writers have attributed some forms of spinal-cord sclerosis to primary arterial affection, and others have noted endarteritis as a coincident phenomenon in affections of the cord, without going more deeply into the question of the connection between them."

He then quotes *Letulle* and *Martin*, who have published cases where tabes was accompanied by general atheroma, and who mooted the question whether any connection can exist between the vessel-affection and the cord lesion: *Ballet* and *Minor*,[1] who published a case in 1884 of diffuse sclerosis of the lateral and posterior columns, where in the sclerosed portions endo-periarteritis plainly existed; the patient had previously been syphilitic. After that Ordonez had claimed to find in arterial change the primary commencement of nerve degeneration in tabes, Adamkiewicz has more recently[2] reasserted his view, the degenerated tracts in the posterior columns in tabes corresponding to the vessels affected. According to his ideas, the vessel affection in these cases would not act in a direct manner (and in this particular he differs from Ordonez), but indirectly through the medium of the interstitial connective tissue binding these vessels together, serving thus to bring about sclerosis and atrophy of the nerve-tissue. *Rumpf*[3] also, taking note of the thickening of the media and adventitia, considers arterial change in certain cases the primary cause of tabes. *Krauss* too[4] investigated in 13 cases of tabes the condition of the vessels in the posterior columns; in most cases thickening of the vessel-walls existed. No distinction could be drawn between such condition of vessels in tabes of syphilitic and of non-syphilitic origin.

"Affections of the heart, and notably of the aorta and aortic valves are not unfrequent in tabes. *Berger* and *Rosenbach* have published the first seven cases of locomotor ataxy with aortic incompetency. *Letulle* and *Grasset* have also collected some cases exhibiting the same association. *Tessier* thinks[5] that in many cases the aortic lesion is not diagnosed during life, and that, post-mortem, small perforations of the valves are to be found; more recently he has published a case where a patient with initial ataxic symptoms developed, after a long tramp one cold night, an aortic incompetency. *Déjérine*[6] described a case in 1884 which had been taken for one of amyotrophic lateral sclerosis, wherein, post-mortem, multiple patches of sclerosis were found, and in which also periarteritis existed. *Demange*[7] also published a case

[1] 'Archives de Neurologie,' 1884, No. 7.
[2] 'Die Rückenmarksschwindsucht,' 1885.
[3] 'Archiv für Psychiatrie,' Bd. 15, No. 2.
[4] 'Neurologische Centralblatt,' 1885.
[5] 'Gazette hebdomadaire,' 1884, No. 40.
[6] 'Revue de Médecine,' 1884.
[7] 'Ibid.

in that year which was considered one of amyotrophic lateral sclerosis, and in which a disseminated myelitis of vascular origin was found; endo-periarteritis of the vessels of the medulla, with general arterial atheroma, was likewise discovered. This year two cases appeared under his observation, in which there was found, post-mortem, a general atheromatous endo-periarteritis of the cord, and in which, during life, the symptoms of lateral sclerosis had existed. *Marie*[1] also has recently drawn attention to cases cited by *Westphal* of ataxy following acute infectious disorders, and has tabulated cases of multiple sclerosis following typhus, small-pox, erysipelas, pneumonia, measles, scarlatina, whooping-cough, intermittent fever, dysentery, diphtheria, cholera, and puerperal fever." He further speaks of arteritis and periarteritis (probably resulting from bacteria emboli) in their connection with the multiple patches in the brain and spinal cord, to define in conclusion such cases as cerebro-spinal localisations of an infectious vessel lesion. "Acute poliomyelitis, acute encephalitis, and multiple neuritis, by their peculiar symptoms and course, cause us to suspect the influence of infection, wherein the infecting virus is localised in the cerebro-spinal system, just as the virus of cholera is localised in the intestinal tract;" and he quotes cases contributed by *Mulder*, which go far to prove that such a surmise is a correct one.

"While observers then at the present day regard vascular change as the primary anatomical cause of various cord affections, there is but little unanimity when such a cause is assigned to tabes dorsalis. Others, opposing the idea that the initial changes arise from the vessels, consider a primary degeneration of nerve tissue as the anatomical starting-point—the doctrine, however, of vessel change as the primary anatomical cause of tabes is steadily gaining ground. Such being the case, and innumerable instances warrant the adoption of such an opinion, there can be no improbability in the assumption that affections which bring about endo- or periarteritis can cause by a spinal or cerebral localisation an acute or chronic change in the cerebro-spinal system. Among the influences which can occasion such a condition, syphilis stands prominently and undoubtedly at the head; but we also know, and *Duplay*[2] has lately again drawn attention thereto, that rheumatism, gout, chronic alcoholism, malaria and lead-poisoning, can bring about like changes; and it is also manifest that sometimes, after an acute infectious malady, endo-periarteritis of spinal vessels may ensue. It is possible that particular infectious vessel-affections such as these may bring about particular spinal cord lesions, as, for instance, that syphilis mainly induces changes resulting in tabes, and that causes other than syphilitic may bring about other spinal affections.

"But it must be remarked, in the first place, that undoubted cases of tabes can be cited without any precursory syphilitic affection, and that syphilitic and non-syphilitic tabes are indistinguishable, either pathologically or clinically, so that there can be no doubt

[1] 'Progrès médical,' 1884.
[2] 'Archives générales de Médecine, 1885.

that vessel-affections of non-syphilitic origin must also rank as responsible agents in bringing about locomotor ataxy; and also is it well worth considering more closely in how far from the pathological investigations, or from the symptoms of the disorder which we call tabes dorsalis, the localisation in a well-characterised anatomical or physiological manner of a certain poison can be determined." Reviewing then the rise and progress of our knowledge of spinal affections, through the combined aid of pathological research and clinical observation, he more particularly deals with the lesions found in tabes dorsalis. "But," says he, "it is not only to the degeneration of one definite column in the spinal cord that we can assign the lesion of tabes. The almost constant affections of the cerebro-spinal system in cases of tabes are:—

1. Changes in the meninges (so decided sometimes that some writers, *Arndt*, *Waldman*, and others, have endeavoured to show that tabes is preceded by a primary leptomeningitis).
2. Lesions of the posterior columns.
3. Lesions of the posterior roots.
4. Lesions of the grey matter of the cord.
5. Lesions of the cerebral nerves.

"Less constant, though still frequently observed, are:—

1. Changes in the cortex cerebri.
2. Changes in the sympathetic system.[1]
3. Changes in the spinal peripheral nerves."

In tabes, then, there can occur certain definite affections of the brain, spinal cord, and peripheral nerves; in other words, changes in the whole cerebro-spinal system. Various combined lesions also sometimes appear, sometimes of posterior and lateral columns,[2] sometimes of posterior columns and anterior cornua.[3] He further maintains that tabes dorsalis can exist while "the classical '*symptômes tabétiques*' of Charcot," with which the lesion of one definite portion of the posterior columns has been associated, are in abeyance. "Sclerosis of the posterior columns is indeed one of the evidences of this affection, but the diversity of the anatomical changes debars us from looking on this one condition as the pathological substratum of this nervous disorder." Clinical evidence is equally indefinite, the symptoms being neither regular nor constant. "*Fournier*[4] evidences psychical disturbances (loss of memory, change of character and disposition, and intellectual disturbances), especially in syphilitic tabes, as the forerunner of locomotor ataxy; with others, retinal atrophy of many years' standing is instanced as a precursor; some cases show disturbances of vesical functions; others gastric and other crises; others, again, epileptiform attacks as the antecedent symptoms. The absence of knee-jerk, a phenomenon first studied by Erb and Westphal, associated with degeneration of the posterior column, was regarded

[1] Raymond and Artaud
[2] Kahler, Prévost, Raymond, &c.
[3] Charcot, Leyden, &c.
[4] 'L'Encéphale,' 1884.

almost as a pathognomonic sign of the presence of tabes. The value of this as such has of late necessarily declined, now that the investigations of *Bernhard*[1] have taught us that after diphtheria this absence of knee-jerk is commonly observed; while later still, we find the same phenomenon noted in sufferers from chronic alcoholism, and in those in weak and debilitated conditions of the system; and still more recently the publications of *Bouchard*, *Rosenstein*,[2] and *Maschka*[3] have proved that in diabetes the same absence of knee-jerk is observable."

"In conclusion," he remarks, "what likeness can we draw between the pathological lesion and the clinical signs, when we find cases recorded where pains in the legs, anæsthesia, analgesia, Romberg and Westphal's symptom, ataxy, &c., were observed, while on post-mortem an intact spinal cord, with however a widely disseminated degeneration of peripheral nerves, was found (cases of multiple neuritis or neurotabes peripherica—Déjérine, Hirt, Hun, &c.), and where cases of diabetes are cited, which by their multiple nervous disturbances and evident absence of knee-jerk are to be regarded rather as cases of tabes (the urine not having been examined) than as diabetic, especially when we consider that cases do occur in which tabes dorsalis is accompanied by glycosuria (Althaus)? Regarding then the manifold changes met with post-mortem in tabes, the variety of the symptoms, their irregular grouping and inconstant exhibition, I believe it to be more consistent to designate those symptoms which we call tabes dorsalis, and which we have heretofore attributed to a lesion of one definite anatomical tract in the spinal cord as a universal affection of the cerebro-spinal system, and that in no instance can either pathological investigation or clinical evidence justify us in definitely attributing to syphilitic influence alone the lesion we find in the posterior columns of the cord." In conclusion, he formulates the opinions he has advanced above under four heads.

"1. Cerebro-spinal localisation of infectious vessel-lesions plays an important part in the etiology of affections of the brain and spinal cord (with which, too, particularly tabes).

"2. Syphilis undoubtedly leads most frequently to affections of the vessels; and, as tabes is the most frequent of spinal-cord maladies, it is only natural that syphilis should be a frequent precursor of tabes.

"3. That syphilis should be considered as the exclusive precursor of tabes, and no other chronic spinal affection is as unlikely as that excluding the syphilitic cause, no other malady causing vascular affection can pose as the cause of tabes.

"4. The direct reason why infectious vessel-lesions should at one time induce diffuse changes, at other times more or less plainly characterised systematic affections, is at present beyond our comprehension."

[1] Virchow's 'Archiv,' Bd. 99.
[2] 'Berl. Klin. Wochenschrift,' 1885, No. 8.
[3] 'Wiener Medicinsche Presse,' 1885, No. 3.

Reviews and Notices of Books.

L'Encéphale, structure et description iconographique du Cerveau, du Cervelet et du Bulbe. By E. Gavoy.

This work consists of two parts, a series of plates of the human brain, and full explanatory text describing the various plates.

The text commences with a preface by Prof. Vulpian, who speaks in highly eulogistic terms of the importance and usefulness of the work, and commends alike the accuracy of the drawings and the amount of labour that it must have entailed on its author.

The first part of this text is devoted to a short general description of the nervous system, while the second part gives a special description of the grey and white matter of the brain. Under the description of the grey matter are given the cortex—with an account of its different layers and their constituent cells—and the basal ganglia. The white matter is described under the headings of arciform, commissural, and convergent fibres. The white and grey matter of the cerebellum and the medulla are likewise described. About forty-eight pages are taken up with this general and special account of the nervous system, while the rest of the text, over one hundred pages, is devoted to the explanation of the different sections.

The essential part of the work, the atlas, consists of a series of plates of sections of the human brain in different planes. The sections have been drawn by the author the exact size, and his drawings have been reproduced by the process of glyptography by M. Silvestre. Some idea of the magnitude of the work can be gained by the fact that only the thickness of one millimetre intervenes between each section and the succeeding one, and that altogether as many as fifty-five plates are given.

The method employed was as follows:—sections were made, one millimetre thick, by a cerebrotome in three different planes; they were then numbered and transferred to a bath, which did not alter the size or normal colour of the sections, but only made them more transparent, and they were mounted between two plates of glass which were sealed at their edges.

The first series, consisting of thirteen plates, was cut vertically, and from before back, *i.e.* sagitally; the second horizontally through both lobes of the cerebrum; while the third series, twenty-three in number, was made in a vertical transverse direction, *i.e.* frontally.

The drawings are most beautifully done and reflect great credit on the author, and the way in which they have been reproduced by glyptography by M. Silvestre leaves nothing to be desired. One would have liked to know the manner in which the brains were prepared, and the form and construction of the cerebrotome by which the sections were made. The author states that the sections were made when the brain was fresh, immediately after its removal from the cranial cavity, but he does not mention whether the brain was frozen or not, when the sections were made. Each section, as it was cut, was transferred to a bath, which, while it increased the transparency of the sections, did not in any way alter their normal colour or produce any diminution in size. The nature of this bath is, unfortunately, not described; and we think this is an important omission, as it would be very interesting to know by what means the author has been enabled to bring out the direction of the fibres very much more distinctly and definitely than it is possible to see in a section of a fresh unprepared brain. It would here be a fit place to mention that a similar atlas to the present one has been previously brought out in America by Prof. Dalton. This atlas consists of a similar series of sections of the human brain, taken in different planes, but with this great difference: that the brains are frozen, and the sections are photographed without any previous treatment or method being employed to bring out the appearances of the various structures. The difference in the number and appearance of the fibres in the drawings of Dr. Gavoy and in the photographs of Prof. Dalton is very marked, the fibres being more minutely delineated in those of the former, though we cannot but think that the latter must be the more accurate.

Though the drawings are most beautifully executed, we find ourselves unable to agree with some of the statements contained in the second part of the text, or the special description of the grey and white matter. On p. 17, § 3, a description of the converging fibres of the brain is given; and although the description occupies over three large pages, no mention is made of the pyramidal fibres, *i.e.* fibres passing direct from the cortex through the internal capsule to the pyramidal tract in the pons, without being attached to the optic thalamus or corpora striata. All the fibres are described as passing from the cortex to the basal ganglia, under the two heads of cortico-thalamic and cortico-striate. We think that even if the author does not believe in the existence of these fibres, that some mention of them should have been given. We must also take exception to the description of the fibres in the cerebellum on p. 25. It is there stated that the medullated fibres enter the grey matter of the cerebellar cortex, and they lose their medullated sheath, traverse the deep layer, and, reduced to their axis cylinders, they reach the superficial zone and there join the prolongations of the large and small cells. According to this account, the fibres lose their medullated sheath before traversing the deeper or granule layer. This, in our opinion, is certainly contrary to the facts of the case, as by using Weigert's method,

the medullated sheath is seen to be present even as far, superficially, as the molecular layer, and as far as we know, no one has demonstrated the point where the medullated fibres lose their sheath. Although the medullated fibres join the basal side of the Purkinje's cells, no one has satisfactorily traced any of these fibres to the smaller cells of the molecular layer, though according to some observers these cells are joined by the protoplasmic processes of Purkinje's cells, but it is mere conjecture whether these cells are joined by medullated fibres from the granule layer.

The atlas will be of great use to any one wishing to learn the relations of the deeper parts, especially, of the cerebrum when viewed in different planes.

C. E. Beevor, M.D.

Abstracts of British and Foreign Journals.

Neurological Contributions from St. Bartholomew's Hospital Reports, 1874–1884:—

1874.

1. On the Clinical Value of Expression in the Insane. By T. Claye Shaw, M.D.

1875.

2. On Laryngismus. By Samuel Gee, M.D.
3. On Incontrollable Impulse. By T. Claye Shaw, M.D.
4. A Case of Idiopathic Tetanus resulting in Cure. By George Hastings.

1876.

5. On Phrenitis Æstiva. By Samuel Gee, M.D.
6. A Contribution to our Knowledge of the Physics of the Cerebral Cortex. By W. Ainslee Hollis, M.D.
7. The Histology of Certain Forms of Degeneration of the Tissues of the Nerve Centres. By W. Henry Kesteven.
8. Epidemic Cerebro-spinal Meningitis. By Neville Hart, M.B.

1877.

9. Further Histological Researches on the Pathology of the Nervous System. By W. Henry Kesteven.
10. The Structure and Functions of the Olivary Bodies. By W. Henry Kesteven.
11. On Spastic Paraplegia. By Samuel Gee, M.D.
12. A Case of great enlargement of the Pons, Crura Cerebris, and Medulla. By Percy Kidd.
13. A Case of Spasmodic Muscular Rigidity with Idiocy. By John Abercrombie, M.B.

1878.

14. On the Cervical Opisthotonus of Infants. By Samuel Gee, M.D., and Thomas Barlow, M.D.
15. A Case of Paraplegia with Sacral Decubitus. By Samuel West, M.B.
16. Cases of Disease of the Spinal Cord. By J. A. Ormerod, M.B.

1879.

17. A Case of Sclerosis of the Cerebral Cortex, with two other examples of Cerebral Disease in Children. By NORMAN MOORE, M.D.
18. A Case of Cancerous Tumor of the Neck, simulating Myelitis. By J. A. ORMEROD, M.B.
19. An unusual Cause of Hemiplegia. By W. E. STEAVENSON, M.B.

1880.

20. On some Intestinal Lesions in the Insane. By T. CLAYE SHAW, M.D.
21. Diseases of the Nervous System. By SAMUEL GEE, M.D.
22. Some Cases of Intra-cranial Syphilis. By J. A. ORMEROD, M.B.
23. Case of Hystero-Epilepsy, with Hemianæsthesia and Chorea. By WALTER H. JESSOP.
24. Notes of Post-mortem Examinations of Cases of Hæmorrhage within the Cranium. By NORMAN MOORE, M.D.

1881.

25. Diagnostic Symptoms of Tabes Dorsalis, with Cases. By J. A. ORMEROD, M.B.
26. Tendon Reflexes in the later stage of Hemiplegia. By J. A. ORMEROD, M.B.
27. Acute and Chronic Insanity. By T. CLAYE SHAW, M.D.
28. A Case of Pseudo-hypertrophic Muscular Paralysis in an Adult. By PERCY KIDD, M.B.
29. Two Cases of Subacute Anterior Spinal Paralysis, with the Pathological Changes in the Spinal Cord of one of the Cases. By CHARLES A. MORTON.
30. Two Cases of Cerebral Disease. By SAMUEL GEE, M.D.

1882.

31. A Case of Lesion of the Sympathetic Nerve in the Neck. By SAMUEL GEE, M.D., and JOHN ABERCROMBIE, M.D.
32. Two Cases of Hemiatrophia Facialis. By WALTER JESSOP, M.B., and OSWALD BROWNE, M.B.
33. Clinical Cases from Dr. Gee's Wards. By OSWALD BROWNE, M.B., and D'ARCY POWER, M.B.

1883.

34. On Criminal Responsibility in the Insane. By T. CLAYE SHAW, M.D.

35. On a Peculiar Numbness and Paresis of the Hands. By J. A. Ormerod, M.D.
36. Five Cases of Tetanus, with some Remarks on its Pathology. By Anthony A. Bowlby.
37. On Trephining the Skull in Traumatic Epilepsy. By W. J. Walsham.
38. Cases of Ocular Motor Paralysis. By W. J. Collins, M.D.
39. On the Pathology and Treatment of some forms of Headache. By J. Lauder-Brunton, M.D., F.R.S.
40. On a Case of Amyotropic Lateral Sclerosis, with Microscopical Examination of the Nerve-Centres, &c. By R. J. Collyns.

1884.

41. Notes on the later Stages of Hemiplegia. By J. A. Ormerod, M.D.
42. On Degradation of Type in the Insane. By J. Claye Shaw, M.D.
43. A Case of Epilepsy, with Remarks. By Howard H. Tooth, M.B.

2. A table is given of 63 cases, which shows that laryngismus is more common in the first than in the last half of the year. In the first are recorded 58 cases, in the last only 5. The author attributes this to the dampness and chilliness of winter and spring. These conditions favour the diathesis and the paroxysm; the former because the child is left indoors more than is good for it, and the latter because the damp and cold air may have an excitant action through the medium of the skin and lungs.

5. The term Phrenitis is chosen instead of Meningitis, because the inflammatory condition implied by the latter term was wanting. The cases related were 4, two boys and two girls, ages 1 year and 10 months, 4 years, 6 years and 5 months, 6 years and 6 months. The attack took place in very hot weather, but in one case not after exposure to the direct rays of the sun. The invasion followed the exposure within 24 hours. The symptoms were briefly as follows: Vomiting, diarrhœa, and shivering. High fever, 108·5 in one case, coldness of the extremities at the same time. There was marked delirium in three cases, sleeplessness, with picking at the bedclothes, and in the other, convulsions. The author regards delirium and convulsions as being of equal significance in children. Coma follows the delirium. In one case were symptoms pointing to affection of the base of the brain, *i.e.* irregular and infrequent pulse, unequal

pupils, right internal squint, deafness, cervical opisthotonus. In two cases the acute stage ended on the sixth day, but the patient has never completely recovered. One case became permanently deaf; the other went into a state of dementia, which, however, is improving. The other two cases died, one on the first and the other on the eighth day.

8. An account of 14 cases of epidemic cerebro-spinal meningitis. Five cases resulted in recovery. Six cases were under 18 years; the remaining eight were between 20 and 50.

11. Four cases are recorded, having as essential characteristics, constant rigidity of legs, or of legs and arms, which increases on handling the limbs, and disappears under chloroform. Other common characters are: that the lesion is congenital, or begins in early infancy. The ages of the children reported are respectively, 10, 8, 8, 3½ years. That of the four cases, two were males. That no tendency to heredity, neurotic or other, was discovered. The affected muscles act well to faradism, and show little or no atrophy, and no tremors. Intellect and sensation natural, no lesion of micturition. General nutrition good. Weakness of back in all cases. Choreiform movement of face in two. Tendency to convulsions in two cases. One suffered from painful cramps in the legs.

12. A girl, æt. 6½ years, in whom the most prominent symptoms were,—double internal squint, slight ptosis right, food lodged between teeth and cheek on the right side, impaired deglutition, deliberate speech, sight and hearing unaffected, but double optic neuritis. *Post-mortem*, the pons was found to be greatly enlarged, most on the right side; the swelling involved the posterior part of the crura cerebri and the left half of the medulla to the decussation. Right of the nerve compressed. Microscopically, was found great absence of nerve fibres in the tumour, which seemed to be due to a great general overgrowth of neuroglia.

13. A girl, æt. 3 years, quite idiotic, born healthy, symptoms began at 9 months old, and consists of spasmodic rigidity of one or more groups of muscles. The spasms may last a quarter of a minute, and then go off as rapidly as they came. On attempting to stand, the leg enters into a state of rigidity, with equino varus and tendency to cross. No sign of syphilis. Mother's father epileptic.

14. An analysis of 25 cases, 10 male and 15 female, illustrating a disease which affects infants under 2 years of age, and having for its essential sign a holding back of the head. The opisthotonus was not constant in all cases, but occasionally ceased for a time. Associated with it in some of the cases were,—rigidity of the limbs, epileptiform convulsions, hydrocephalus, the latter affection coming on some time after the opisthotonus was established. In 11 cases the disease was chronic, and termination unknown, but of the remaining 14, only 3 recovered. As to post-mortem appearances, no tubercles were found in the brain or membrane, but basic meningitis in all cases. In five cases the disease had existed from birth, two were certainly syphilitic, another probably so. Some of the cases resembled epidemic cerebro-spinal meningitis. There was no evidence of heredity, either ancestral or collateral. In three cases was a history of a fall before the onset of the disease, but fracture or laceration of brain or membrane was found after death. In two cases there was otitis interna.

17. These cases are related at considerable length and illustrated by woodcuts. The first was that of a child, aged 5 years. Her history was briefly, that after a fright she was affected by uncontrollable facial contortions on the left side, followed by left hemiplegia. Later, there supervened right hemiplegia with complete loss of speech. The right hemiplegia improved, but in about five weeks great contracture of the left limbs set in. There was slight hyperæmia of the optic discs. *Post-mortem* was found yellow softening of (1) the hinder part of the first frontal convolution; (2) the hinder part of the three frontal convolutions; (3) ascending parietal, and (4) ascending frontal convolutions; (5) callosa marginal convolution as far as the fissure of Rolando. This softening was symmetrical on both sides of the brain, except that it extended in the frontal region nearer to the olfactory sulcus on the right than on the left side. The vessels at the base were reported quite natural. The other cases described were,—one cerebro-spinal meningitis, the other of acute hydrocephalus, with accounts of autopsies in each.

21. Illustrated by the following cases: (1) Spastic paraplegia in infant and adult age. (2) Spastic diplegia. (3) Sclerosis of encephalon in inherited syphilis. (4) Paralysis due to compression of arm. (5) Traumatic myelitis, followed by muscular atrophy, (?) spastic rigidity. (6) Motor ataxia of legs, with paralysis of orbicularii oris. (7) Teichopsia. (8) Infantile spinal paralysis.

22. A careful record of three cases, among the symptoms of which are,—headache, more or less severe; drowsiness, eye symptoms, paresis and anæsthesia of certain limbs, epileptiform seizures. All three cases improved greatly under iodide of potassium and mercury.

25. An account of 21 cases of tabes dorsalis, arranged in three series: (1) Cases in which there was well-marked inco-ordination in the legs, causing difficulty in walking and inability to stand with the eyes shut. (2) Cases in which such inco-ordination was slightly marked. (3) Cases in which there was no inco-ordination. The relations of the inco-ordination to the pupil affections are also carefully discussed.

26. A suggestive paper showing the relation of the tendon-reflexes to rigidity in hemiplegia. The author finds that the knee-jerk in some cases of hemiplegia, even a long time after the attack, is the same on the affected as on the sound side. In such cases late rigidity rarely or never occurs. On the other hand, if the tendon-reflexes be increased on the affected side, rigidity may be predicted with comparative certainty. These points are well illustrated by tables comprising 50 cases of hemiplegia.

28. Man, aged 24; the calves had been noticed to be large for a very long time, but the more essential symptoms dated back about three years. A younger brother had been complaining of difficulty in running, but no enlargement of the legs had been observed.

Arms.—Triceps both enlarged. Biceps and flexors of forearm rather large. Pectorales and latissimi dorsi much wasted. Deltoids, infra- and supra-spinati, masseters, temporals, thumb-muscles and tongue, unaffected.

Legs.—Glutæi and thigh-muscles much wasted, except vasti externi. Calf-muscles much hypertrophied. Faradic irritability of hypertrophied and wasted muscles slightly diminished. Knee-jerk very feeble. This case is interesting from the fact that it is probably an example of Erb's recently described "Juvenile form of Progressive Muscular Atrophy."

30. (1.) *Hemichorea.*—A painter, æt. 54, no lead line or symptom of plumbism. Distinctly gouty. History of patient: Choreic movements of right hand five days before admission, soon spreading to leg. Never on left side. Died thirteen days after admission. In the left internal capsule, close to the thalamus at the junction of

its anterior and middle thirds, and separated from the lenticular nucleus by a band of healthy white matter, was a small patch of softened brain of the size of a lentil, and of an ochre hue. Also in the posterior half of the right thalamus was a small reddish cyst, the size of a millet-seed. The author emphasizes the following points: (1) That it was a case of simple chorea. (2) The onset was sudden, but there was no apoplectic symptoms. (3) No antecedent palsy. (4) No hemianæsthesia. (5) Cranial nerves unaffected. (6) The lesion was in the direct pyramidal tract. In cases of hemichorea associated with hemianæsthesia, the lesion has been found *behind* the pyramidal tract. (7) Probably embolic.

2. A second case of gelatiniform enlargement of the Pons Varolii.

31. Among the earliest symptoms was drooping of the right upper eyelid, closely followed by paralysis of the right side of the body. Right pupil half the diameter of the left, and does not dilate as fully with atropine as the left. Right forearm flabby, but with fair movement; can hardly move fingers at all. Faradic irritability of right superficial, and deep flexure of hand, pron. rad. teres, thenar and hypothenar, and interossei, abolished. Paralysis of both legs followed later, with absence of tendon reaction, superficial reflexes, and with complete anæsthesia. Patient perspired much about the head during sleep, and twice it was noticed that the perspiration was confined to left side of the head. Autopsy discovered a sarcoma connected with the first and third ribs, spreading into the spinal canal, and pressing on the cord. The right sympathetic was involved in the growth.

32. Two interesting cases, one of a girl, æt. 12, the other a woman, æt. 48. Illustrated by two photographs. In both, the atrophy made its appearance at an early age, and was associated with ocular troubles. In the second case it was preceded by intense pain in the side of the head. In both, the left side of the face was affected, and was marked by extreme atrophy of soft parts, with wrinkling of skin, loss of hair, and some wasting of the bones. In the child there was wasting of the muscles of the left arm, pectoralis major, deltoid and infra spinatus, with great depression of skin in palm, with wasting of middle and ring fingers. In both cases the muscles react well to faradism. Sensation is normal.

Mr. Jessop discusses the various theories of causation of this affection, and suggests that lesion of the Gasserian ganglion might

account for the facts. The known connection of the sympathetic with the ganglion might have to do with the ocular affection.

33. These cases include, (1) a case of locomotor ataxia, associated with perforating ulcer of the foot; (2) a case of subacute anterior general and spinal paralysis; (3) a case of hereditary locomotor ataxia.

35. This article is illustrated by twelve cases, all women over 40 years of age except three. The symptoms always commence at night. The patient wakes up with numbness and formication, and even pain, in the hands or arms. There may be also some loss of power. Symptoms pass off after friction of the part, and much resemble that state induced by compression of the nerve-trunks. The affection, however, is often bilateral. The author compares it rather with a mild case of tetany, without the facial irritability. The use of bromide of potassium was attended with the best results.

36. In four cases of tetanus, Mr. Bowlby was able to make a careful post-mortem examination. In all, the nerves round about the wound were found to be perfectly healthy; the same may be said of the spinal cord, medulla, pons, corpora striata, and optic thalami, except in one case in which there was softening of the cerebrum just above the left Rolandic fissure, also in both corpora striata. In no case was the blood found to contain micro-organism.

The author summarises the current theories, and proceeds to show how baseless these theories are, both from pathological and clinical grounds. He himself inclines to the view that the disease is essentially one of blood-poisoning, in support of which he adduces these facts: (1) That the occasional idiopathic occurrence of the disease shows that local injuries are not essential for its production. (2) Its extreme prevalence in certain climates and localities. (3) Its preference for coloured races rather than white. (4) Its occasional appearance in epidemic form. The constitutional origin of the disease is shown strongly in the form called tetanus neonatorum, which attacks the children of coloured parents; the umbilical cord is always in an unhealthy condition, and bad hygienic surroundings always play some part in the affection. Here the peripheral nerve-theory falls to the ground, as there are no spinal nerves in the umbilical cord.

37. To this paper, which is principally of purely surgical interest,

is appended a series of tables comprising cases collected from all sources, European and American. They contain a record of 82 cases, in which most of the details are forthcoming, and 48 of which no details are recorded, making, in all, 130 cases, with a reference to each. In 74 cases complete recovery followed. In 18 cases improvement followed the operation. In 7 cases no improvement, and 30 cases died.

39. In enumerating the causes of headache, the author brings them under two heads: (1) Local irritation. (2) Causes having their origin in general conditions. Under the first head come decayed teeth; abnormalities of refraction in the eye; diseases of the ear, nose, throat, &c. Under the second head come indigestion, biliousness, constipation, fever, plethora, anæmia, rheumatism, gout, and albuminaria. He makes some interesting remarks on the position of the pain. Thus, in caries of the lower molar teeth, the headache is usually in the occipital region; decay of the upper molar causes temporal headache; while frontal or vertical headache may be associated with decay of the incisors and canines. The headache of anæmia is generally vertical. Frontal headache, with constipation, is relieved by salines; while that without constipation is best treated with acids or alkalis.

40. Full details are given of a case of a man aged 32, with no personal or family antecedents of any importance. The first symptoms consisted of wasting of the left-hand muscles, followed quickly by wasting of right hand and both arms, and at the same time dysphagia and other bulbar symptoms made their appearance. The paralysis, rigidity, and tremor proper to lateral sclerosis were of later occurrence. A full account of the post-mortem appearances, gross and minute, of the cord, nerves and muscles is given.

The author considers that the grey matter was primarily affected, the sclerosis spreading from this to the anterior columns and crossed pyramidal tracts.

41. Short descriptions of 20 cases of hemiplegia due to simple non-progressive cerebral lesion. The cases are arranged to elucidate the following points:—

(1.) Two cases to show that, contrary to the rule, the tendon phenomena are sometimes exaggerated without the supervention of contracture, and from cases in which they are more marked than on the sound side.

(2.) The tendon reaction is sometimes found exaggerated on both

sides, and a case is quoted in which the paralysis is found on the same side as the lesion as well as on the opposite side. The author discusses the explanation of this post-hemiplegic paraplegia, and then describes,

(3.) Two cases of incontinence of urine after hemiplegia.

(4.) A case of hemiplegia, to which was superadded degeneration of the anterior horns of the grey matter, shown by rigidity, atrophy of thumb-muscles, and marked degenerative reaction.

(5.) Two cases of rigidity of hand and leg, alternated with involuntary slow movements ("mobile spasm"), which are easily provoked by any attempt to move the rigid limb.

(6.) Eight cases of post-hemiplegic epilepsy. In these the fits may occur at any age, and appear to have some direct relation to the hemiplegia, but do not make their appearance till some time after its occurrence.

HOWARD H. TOOTH, M.B.

A Study of the Disturbances of Co-ordination in Children. By A. ERLENMEYER, M.D.—The case reported is that of a boy, the only child of related parents (first-cousins). He had the gait of locomotor ataxy, and restless movements of the arm. It is noted that he was seen by the late Professor Friedreich and Professor Erb: the former diagnosed spinal disease; the latter, chorea minor.

History.—Well till the age of 5, then a fever of uncertain nature (? cerebro-spinal meningitis); five months afterwards the following nervous symptoms developed very rapidly—unsteadiness of gait, restless movements of arms, transitory tremulousness of the eyeballs. On admission (three years afterwards), there was well-marked tabetic gait, inability to stand with eyes shut, absence of patellar tendon-reflex: constant irregular movement of the arms, and (in connection with them ?) of the trunk and head; movements of the facial muscles and right lower eyelid; right internal squint; right pupil larger than left; both pupils acting normally. Speech described as "indefinite, difficult, showing irregularities in the mechanism of word-formations." No nystagmus. No affection of sensibility. The squint disappeared under treatment; the speech improved; but in other respects the boy remained in the same condition.

The author looks on the case as one of hereditary ataxia (locomotor and static), of which the fever was the determining cause.

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